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CONTENTS

		PAGE
Original Papers		
Cerebrospinal Fluid examined by Ultra-Violet Light	FRETSON SKINNER	97
The Physiological Basis of Repression and Dissociation	R. G. GORDON	105
A Neurological Test for Stutterers	ROBERT WEST	114
Critical Review		
The Nature and Significance of Senile Plaques	MACDONALD CRITCHLEY	124
Editorial		
Neurosyphilitic Uncertainties.		140
Abstracts		144
Reviews and Notices of Books		180
Occasional Notes		191

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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY.

Vol. X. OCTOBER, 1929. No. 38.

Original Papers.

CEREBROSPINAL FLUID EXAMINED BY ULTRA-VIOLET LIGHT.

By

E. FRETSON SKINNER, Sheffield.

During the course of some examinations of cerebrospinal fluid in cases of chorea the idea suggested itself that some form of physical examination of the fluid might give interesting results.

Cerebrospinal fluid contains, amongst other things, colloid bodies, and under certain pathological conditions the amount of these colloids and probably the kind of colloid seem to vary. Such variation is demonstrated by the ordinary gold test. This test probably depends on precipitation of metallic gold from a colloidal solution by changes in the electrical charges on the surface of colloidal particles, and as it seemed possible that the interposition of cerebrospinal fluid in the beam of the spectrum might show some absorption such as is seen in the well-known hæmoglobin bands, a series of observations was planned to ascertain whether such absorption would occur. I may say at the outset that the method has not realised this hope and my only excuse for recording these observations is that there is some slight indication that, at times, in certain conditions, there appears to be some clinical application.

All known forms of radiant energy are probably due to wave motion in the ether, various forms of energy differing only in wave length. If we represent diagramatically all these forms of energy by a horizontal series of vertical lines, each one of which corresponds to a definite wave length, we obtain a long band such as is indicated in the diagram below (Fig. 1.*).

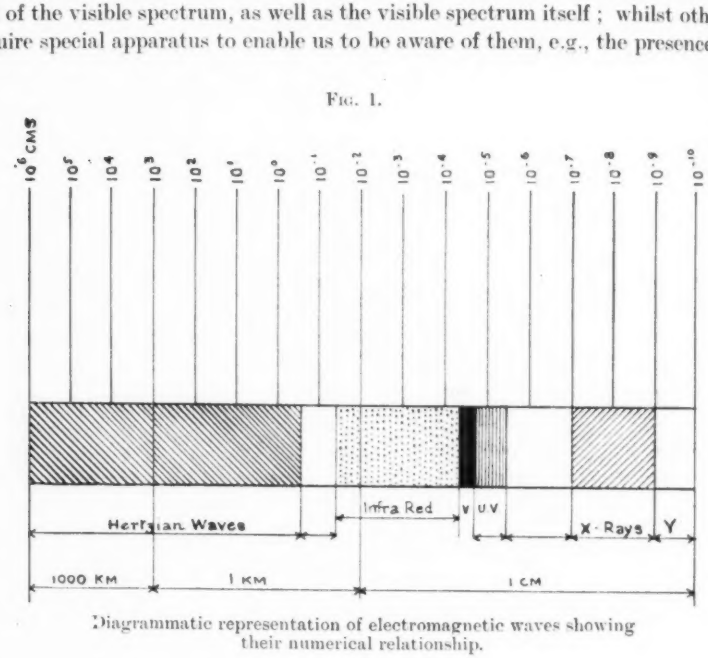
From this diagram it will be seen that the differences in wave length are enormous. If all the waves between the visible spectrum and the gamma rays of radium were represented on this diagram by the horizontal space of 1 cm., in order to present all the other wave lengths a horizontal band of about

*Copied from "X-Ray" by Schall, p. 54.

the width of the Atlantic Ocean would be required. In the diagram the infra-red portion has been enormously shortened, whilst the ultra-violet portion has been similarly lengthened. The actual wave lengths of these various forms of energy vary from 35,000 metres (20 miles approx.) to a "tenthmetre," which is one ten-thousand millionth of a metre.

These various waves can be appreciated in various ways, some of them by means of our unaided senses, for example some of the heat waves below the red end of the visible spectrum, as well as the visible spectrum itself; whilst others require special apparatus to enable us to be aware of them, e.g., the presence of

FIG. 1.



Diagrammatic representation of electromagnetic waves showing their numerical relationship.

ultra-violet waves can be detected by their action on a photographic plate, the Hertzian waves by means of the thermionic valve, etc. But wherever these wave-forms occur in the band of radiant energy, they are all the result of some force which has set in action vibrations with a definite periodicity.

The fragment of this vast series of wave-like forms of energy with which I am going to deal is one which comprises wave lengths varying from 7,594 Angstrom units at the red end of the visible spectrum to 2,149 Angstrom units at the end of the ultra-violet band.

If a beam of monochromatic light be passed through the narrow slit of a spectroscope a brilliantly coloured image of this slit is seen on the screen; for example, if sodium chloride is burned in the non-luminous flame of the Bunsen burner two brillant yellow images of the slit are formed, due to the

monochromatic light emitted from sodium vapour. Similarly a salt of thallium will produce a bright green image, but this green image will be in a different situation from that of the yellow sodium images, and by appropriate means the positions of these bright images can be shown to be fixed in the band of the visible spectrum. Almost all the elements can be made to produce luminous coloured bands on the spectroscope screen, which bands correspond to definite wave lengths—in other words, the wave length determines the colour of the image seen.

Kirchoff (1859) showed that if the rays from a source of intense white light which consists of innumerable waves of monochromatic light, such as that produced by a carbon arc, were passed through the spectroscope, the usual prismatic colour spectrum was produced, but if a Bunsen burner with sodium chloride burning in it were interposed between the carbon arc and slit of the spectroscope there appeared in the colour spectrum two dark lines in the neighbourhood of the yellow bands, and these were found to be identical in situation with the luminous images given by sodium vapour.

Kirchoff explained the phenomenon by stating that the amount of light proceeding from the sodium vapour is relatively so small that the only effect to be considered is that which the presence of this vapour may have on the rays proceeding from the white hot body of the carbon arc. The ether waves proceeding from this white hot body will pass readily through the sodium vapour, except those vibrations whose wave lengths are the same as those of the screen of sodium vapour which absorbs them.

This simple experiment explains the Fraunhofer lines in the solar spectrum. The body of the sun is so much hotter than the surrounding incandescent vapour (photosphere) that the rays of light pass through a relatively cooler vapour in which are various elements whose vibratory periods are the same as some of the waves coming from the sun's body, and which, consequently, absorb those particular waves, producing black lines on the solar spectrum.

This brings me to the reason of my paper. Absorption spectra are merely spectra in which certain waves are damped or absorbed by some substance through which they have to pass.

The various absorption spectra of hæmoglobin and its derivatives are all familiar, and since absorption bands are so very definite and so mathematically constant I thought it would be of interest to see whether cerebrospinal fluid would give any such bands. When it is remembered that the number of leucocytes may vary in the cerebrospinal fluid from one to eight hundred per c.mm. or more, and that the constituent colloids may show very considerable variations, as may also the salts, it seemed possible that some variation in the spectrum might occur if cerebrospinal fluid were interposed in the path of the rays of light passing into a spectroscope.

The recording of spectra on photographic plates depends on the action of ultra-violet rays, so it was necessary to choose as an emitting body some

substance which, when raised to a white heat, would give off ultra-violet radiations. The copper arc was chosen because it is fairly rich in ultra-violet rays and possesses a number of very clearly defined lines in its spectrum. Tungsten is richer in ultra-violet rays but it is very expensive and burns away very quickly, whilst iron has so many fine lines that they are difficult to calibrate.

The apparatus I used was very simple, consisting of a copper arc, enclosed in a light-proof box, a small quartz cell for containing a few c.c. of cerebro-spinal fluid, and a spectrometer for analysing the light rays and recording them photographically.

As I stated previously, I have been disappointed in not finding any selective absorption in the spectra I have examined, but such results as have been obtained are described below.

In the first place, Fig. 2 represents the normal spectrum of incandescent copper in which it will be seen there are a large number of very clearly defined lines whose wave lengths are known and which can be easily identified. It comprises a series of wave lengths varying in magnitude from 5,782 Angstrom units to 2149 Angstrom units; that is, it begins at and includes the visible spectrum and a considerable amount of the ultra-violet band.

FIG. 2.

The next two figures (Figs. 3 and 4) show the amount of absorption which occurs when ultra-violet rays are passed through distilled water and normal saline respectively, and it will be seen that the amount of absorption is very slight, occurring at the right-hand of the band.

FIG. 3.

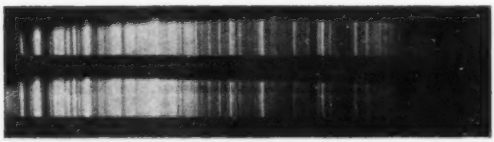
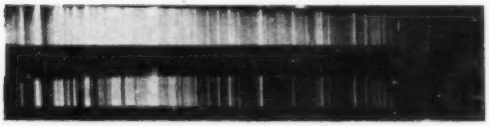


FIG. 4.

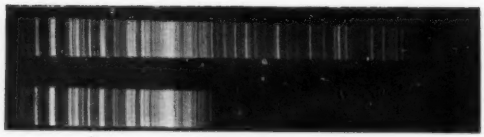


It was necessary in the first place to examine a number of normal lumbar fluids, in order to ascertain what kind of absorption occurred. It is not easy to obtain normal cerebrospinal fluid since lumbar puncture, though a simple procedure, is not without a slight risk of grave consequences, so that it is not undertaken unless there is an indication for it, in which case the cerebrospinal fluid cannot be considered normal. However, I have managed to obtain several samples from individuals who volunteered and who, so far as I could ascertain, were absolutely healthy. These fluids were examined with various arcs and they all showed the same type of result, general absorption occurring about two-thirds of the way down the band. Fig. 5 shows the normal spectrogram of cerebrospinal fluid, using copper to produce the ultra-violet rays.

FIG. 5.

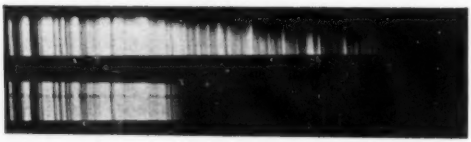
The most striking changes in cerebrospinal fluid that one meets with in ordinary routine clinical work are perhaps found in cases of general paralysis. In these cases the fluid often contains a very large number of leucocytes as well as a considerable increase in the amount of proteins present, and the colloidal gold test is usually very definite. In view of these striking changes some cases of general paralysis were first of all examined and after a large number had been submitted to the test it appeared that very often a fairly definite spectrogram was obtained. This spectrogram is shown in Fig. 6, in which it will be seen that complete absorption of the rays occurs below a wave length whose value is 2,961 Angstrom units, with the exception of one well-marked band which corresponds to a wave length of 2,618.

FIG. 6.



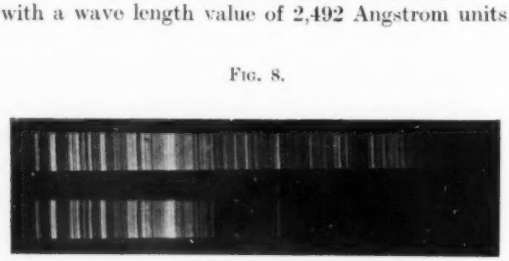
In certain cases of advanced general paralysis even this band, 2,618, disappears. Such a spectrogram is shown in Fig. 7.

FIG. 7.



The next series of fluids to be described were taken from cases of tabes, of which Fig. 8 is an example, where it will be seen that absorption is not so complete as is the case in fluids obtained from general paralytics, one other band being visible with a wave length value of 2,492 Angstrom units.

FIG. 8.



But here again in old-standing cases of tabes the spectrogram may be identical with that of general paralysis, and one is tempted to suggest that the absorption of tabetic fluids is an early stage of that given by general paralytics.

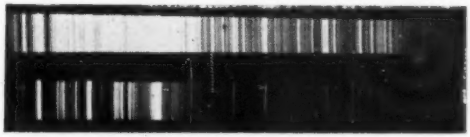
The most striking of all these spectrograms is given by fluids from cases of tuberculous meningitis. This is not a particularly common condition and I have not been able to examine a large number but, thanks to the courtesy of colleagues, I have obtained fluids from perhaps a dozen cases; all of these show rather a characteristic picture which is illustrated in Fig. 9, in which it will be seen that there is complete absorption from a wave length of 3,248 Angstrom units.

FIG. 9.

Although the number of cases so examined has been small I am inclined to think that this picture has some diagnostic significance, in support of which I might cite the following clinical case.

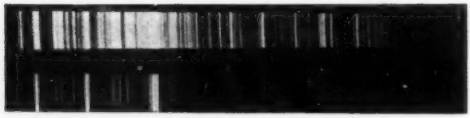
One afternoon, whilst working at these spectrograms, I was informed that a case of tuberculous meningitis had just been admitted to hospital and a lumbar puncture was being done. Some of the fluid was sent down to the laboratory in the ordinary way and some was sent to me for examination. I took a spectrogram and on examining the negative I felt sure that the case, which I had not then seen clinically, was not one of tuberculous meningitis. Going into the laboratory later I found the routine examination of the fluid had been carried out and the pathological opinion of tuberculous meningitis had been expressed on the grounds of a very considerable diminution in the chloride content. This was the only pathological change, as there was no increase in cells nor was the gold test positive. Next day I saw the child and clinically it certainly appeared to be a case of advanced meningitis, with head retraction, Kernig's sign and so on, and I must confess I was considerably shaken in my opinion. None the less I maintained it and within a few days it was obvious that the case was one of so-called meningismus and not true meningitis, since all the meningitic symptoms gradually abated and the child made an uninterrupted recovery. Fig. 10 shows this particular spectrogram.

FIG. 10.



Similarly, Fig. 11 is from a case in which the diagnosis of tuberculous meningitis was made on clinical grounds but which eventually determined itself to be a meningismus complicating a lobar pneumonia in a girl eighteen years of age.

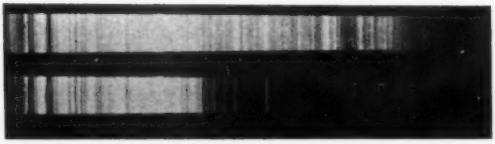
Fig. 11.



The conditions I have cited seem to give spectrograms which appear more or less constant in type but on what they depend at present I cannot say. I am quite sure they do not depend on the number of cells present in the fluids, nor do the variations in the protein constituents appear to bear any fixed relationship. The only definite statement possible is that absorption seems to take place in toxi-infective conditions and varies directly with the severity of this process ; the nearer a fatal termination the more complete the absorption.

I have examined cerebrospinal fluids from a number of other clinical conditions but none of them produced such definite pictures as those I have mentioned. Disseminated sclerosis shows some absorption, but not nearly as much as in tabes, and of all the organic nervous diseases most nearly approaches that of normal cerebrospinal fluid (Fig. 12.).

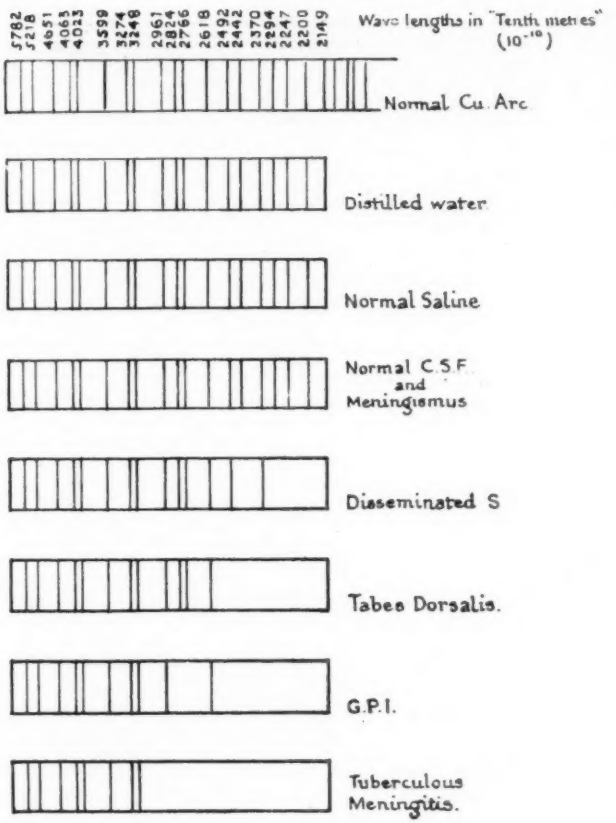
Fig. 12.



I have tried to epitomise these results in the diagram (Fig. 13) but do not wish to present any claims for real usefulness for these spectrograms at present ; there are so many pitfalls and possibilities of error that I should not like anyone to suppose that I had advocated a new diagnostic procedure. But, at the same time, I think there are certain suggestive points in the observations which warrant a continuance of the experiments with more careful calibration and a more rigid technique, which I propose to do in the near future.

I should like to express my thanks to those of my colleagues who have provided me with fluids for examination ; to Professor Milner of the University for permission to work in the Physics laboratory and to Dr. J. N. Clarke for much extremely valuable expert advice and help.

Fig. 13.



Schema of absorbtion spectra of various cerebrospinal fluids.

THE PHYSIOLOGICAL BASIS OF REPRESSION AND DISSOCIATION.

By

R. G. GORDON, Bath.

Most of those who are interested in psychopathology are prepared to admit that repression and dissociation are processes which underlie many of the symptoms of psychoneuroses and psychoses. We may, however go further than this, for we may see the same processes at work in the normal individual. Since none of us are perfectly adjusted either in respect of the interplay of our own impulses and emotions or in our relation to the environment, we have to make use of some means of reducing or overcoming the friction produced by this maladjustment, and repression and dissociation are two of the commonest means whereby this avoidance of friction is achieved. We are all familiar with the tendency to deny the existence of certain events or impulses and the logic-tight compartments built round incompatible constellations. It is our object to find an explanation of these two functions which accords more with physiological principles than do many of those advanced by the newer schools of psychopathology. Many presentations of Freud's teaching seem to bear too anthropomorphic an appearance, describing as they do the field of consciousness with a censorship dividing it from an unconscious realm containing repressed and dissociated material. Freud's own concept of the censor, however, was formulated simply as a convenient means of description and he deliberately left any physiological explanation on one side; his description of the censor is of a barrier and nothing more.

If we are to obtain any conception of what we mean by the terms 'repression' and 'dissociation' from the physiological standpoint, we must try to find a common ground of expression between physiology and psychology. I believe that this can be discovered in the concept of a strict correlation between patterns of behaviour and patterns of neuronic activity. I think that everyone will agree that there can be no expression of mind *as we know it* without the activity of brain; and that no activation of brain takes place without some phenomena which may be objectively recorded by an observer and/or subjectively experienced by the individual concerned, and which may legitimately be called mental.

The acceptance of such a postulate does not commit us in any way to accepting any theory as to causality; we do not say that mental activity is nothing but a phenomenon of cerebral activity on the one hand, or that the psychological situation determines the activation of a certain group of neurones on the other. We do not even concern ourselves whether mental and cerebral activity is interdependent or independent. These are subjects which may safely be left to the metaphysicians.

At low levels we are content to regard the observed effects as correlated with the activity of neurones which we can actually recognize and map out. This is true of the various reflexes whose paths through the nervous system have recently been described with an increasing degree of accuracy. At such levels we are not accustomed to describe any 'mental effects,' but it does not do to be too dogmatic as to the absence of such effects. This applies more especially to reflexes in the vegetative field, for who shall say how much such reflex actions contribute to general coenæsthesia, which is an important factor in mental situations? As we get higher in the scale it becomes less and less possible to trace out the actual neuronic paths, but we can still envisage the locality of various functions. We know the importance of the structures in the floor of the fourth ventricle in relation to vital function and the regulation of the vegetative organs. We now recognize the importance of the interbrain and more particularly the subthalamic region in relation to primitive affective and more especially painful reactions. To quote Tilney: "Clinical evidence seems to be insistent that the thalamus is a centre for affective tone. Its irritation or destruction leads to changes in affective expressions and attitudes. The thalamus is a part which when diseased may lead to forced laughing and crying. It is a primary centre for involuntary movements and emotional expression. Psychic processes representing a certain degree of consciousness may be carried on by the thalamus independent of the cerebral cortex. These processes are probably limited to painful sensations. The thalamic centres are chiefly concerned with affective experience. Destructive lesions which involve the cortex alone do not necessarily disturb the painful and affective qualities of sensibility." Such functions are of course co-ordinated, discriminated and controlled through their connexions with the cortex. "Personality and behaviour in their most complex form still retain their relations to the primitive emotions. The archaic current of feeling tone arising from the interbrain pervades all the higher psychic faculties and colours them with some degree of pleasure or displeasure."

A good deal of more or less precise knowledge exists as to the localization of function in relation to discriminated and controlled motor activity, while the areas of sensory radiations which are concerned with more purely cognitive mental activity are also delineated with some exactitude. Pavlov's work on conditioned reflexes shows how various sensory and motor functions are analysed in the cortex and how inhibitions may disturb the chain of conditioned responses under certain conditions of cortical activity. The integration of cognitive functions with feeling which may result in what Tilney calls motor incentive to co-ordinated action would on anatomical grounds seem to take place in the frontal lobe, but it may prove on further investigation that the final co-ordinating and discriminating functions of the cortex depend rather on the more recently developed layers of cells than on the more recently developed areas. It thus appears that it does not require any very remarkable effort

of imagination to correlate a neuronic pattern with even the most complicated pattern of psychological significance. In any psychological constellation leading to a pattern of behaviour of whatever kind, we may picture the activation of a complex pattern of neurones extending from the spinal cord through the hindbrain, the interbrain, and the general cortex up to the frontal and prefrontal regions and the superficial layers of most recently added grey matter. If we do not strain the analogy too far we might liken such a neuronic pattern to a series of railway lines with all the points set right, so that the traveller may pass over just that series without delay or difficulty. It goes without saying that the more habitual such a constellated pattern of behaviour becomes the more set do the points become, the more easily does the traveller accomplish this particular journey and the more difficult it is for him to branch off on to other directions.

It is obvious that it is our purpose here to correlate such more or less closed patterns of neuronic activity with the constellations of ordinary psychology and the complexes of analytic psychology. It has been objected that such a correlation makes no allowance for the undoubted dynamic quality of the complex in psychic behaviour. I do not believe this is a valid objection if due regard is had to the significance of form. How universally important this significance of form is, is a commonplace of modern knowledge. The discoveries of twentieth century physics have deposed substance and enthroned form, while æsthetic has of recent years concerned itself to an increasing extent with this same subject of form and pointed out its universal significance. Dynamic efficiency depends not only on the *vis a tergo* but also on the absence of friction. Energy must of course originate from katabolic processes, and the amount of available energy may vary in different parts of the body or even of the nervous system. Of such matters we are at present ignorant, and we do not know whether one action which appears more forceful than another owes this property to its own energy or to lack of external obstructions. After all, the neuronic pattern is held to be no more than a track for the spread of activation; and if this activation proceeds along a track of a particular form and of no other form this may have the most profound influence on subsequent behaviour. If activation is for example prevented from leaving this pattern form so that it cannot spread to other patterns, we can see how such intensely compulsive reiterations as are found in the obsessional neuroses and similar conditions may arise. We may thus envisage repressed or dissociated complexes which exhibit the tripartite characteristics of cognition, affection and conation as being correlated with an activity spreading through a neuronic pattern involving neurones in the spinal cord, the hindbrain, midbrain and cortex. Such activations may be expressed as activities of the vegetative organs, feelings organized as emotions or disorganized as angoisse and similar manifestations, thoughts practical and phantastic, and actions simple or complex.

If this presentation of the physiological correlate of the psychological concept is accepted, in order to demonstrate our theory of repression it is

necessary to consider another important function of the nervous system. Sherrington has shown how the nervous system as a whole exercises an integrative action, and has formulated the principle of reciprocal innervation. Under ordinary circumstances we find that these reciprocal actions are reasonably harmonious, but become less harmonious as we ascend the scale of nervous levels. The perfect reciprocal action between agonist and antagonist in the sphere of voluntary muscular activity would be a source of continuous wonder were it not such a matter of course. In the vegetative field this is not always so much a matter of course; but in health the ordinary integration of, e.g. contraction and relaxation during the process of peristaltic action of the bowel is so smoothly carried out that we are unconscious of our digestive function. When we come to the emotional field we find less perfect integration in our daily life. Both in animal and human behaviour it is a commonplace to observe the indecisive actions due to a conflict between curiosity and fear, between fear and anger, or between the more complex patterns which we call self-assertion and self-abasement.

When we come to examine pathological conditions in which this integration has broken down we find a state of affairs which I submit throws considerable light on the subject of repression. In the sphere of voluntary muscular action let us consider that form of hysterical paralysis which was so common during the war and which is by no means rare even in these days of peace. The limb is held rigid, the flexors and extensors both being in action at once, the action of one group in fact cancelling out the action of the other more or less completely. Close observation showed that if this mutual cancellation was exact and complete the limb was held rigid and still; but if, as was usually the case, it was not exact, minute movements in either direction were permitted. This phenomenon is generally described as tremor, which in these cases might be regular, fine, or gross according as the action of either group escaped from the lock imposed by the other, regularly or irregularly, for an instant or for longer; but since this movement is physiologically distinct from the tremor of organic disease it would be better to describe it as oscillation. In the vegetative field the principle of reciprocal innervation is the same, though the time relationship is different; for while voluntary muscular integration requires a relaxation of antagonist synchronously with the contraction of the agonist, in involuntary muscular action the relaxation precedes contraction in time so that the peristatlic wave results. In addition to transitory disturbances of this rhythm there is evidence to support the theory that such conditions as achalasia gastrica and Hirschsprung's disease are due to a disturbance of this integrative function and not to any structural obstruction. When we observe vegetative function as a whole and consider the physical accompaniments of anxiety as described by the term 'angoisse' do we not see an oscillation analogous to the tremor or oscillation accompanying hysterical paralysis? Here there is an incomplete balance of vagal and sympathetic function so that various symptoms occur

at one time characteristic of vagotonia and at another of sympathicotonia, much to the discomfort of the patient. Thus we may find a rapid pulse, a certain degree of bronchial spasm, a dry skin, an atonic bowel as present together—to the confusion of those who would try to discover a tonus of one sort or another, but explicable on the theory of oscillation.

In the emotional sphere such cancellations and oscillation effects are met with even in normal life, but still more in pathological conditions. As McDougall has pointed out in discussing the schizophrenic, sulking is an example of the cancellation of opposition. He says, "The sulking child (or adult) is one whose self-feelings are wounded; his self-assertive tendency has been thwarted, he broods over the insult or injury; but he can neither assert himself energetically in new efforts, nor frankly accept the rebuff and yield to superior force; unable to accept a rebuff with deference, or respect, or admiration, he withdraws from all social contact and broods, a prey to a painful conflict in the very citadel of his personality, his sentiment of self-regard; all interest in the outer world arrested, all outer activity paralysed."

"Embarrassment is a fleeting though painful conflict between the self-regarding affects; sulking is a deadlock between them. And sulking is a dangerous condition, already half-way to disorder. In children it normally terminates with the victory of one or other impulse, with an outbreak of naughtiness and violent self-assertion, or in a scene of reconciliation in which the child sobs on his mother's shoulder and promises to be good. In the adult, sulking is apt to terminate more tragically than in the child. The sulking, the self-absorbed brooding over slights or insults, may terminate in an outbreak of vengeful violence or; as in the introverted Malay, who after sulking (like Achilles in his tent) takes his kris and runs amok, cutting down every human being he meets, until he is himself cut down. I am suggesting that the schizophrenic state is essentially a morbidly exaggerated and prolonged state of sulking; and that the sudden outbreaks of violence which such patients are liable to, even those that seem utterly apathetic, are strictly comparable to the amok in which the sulking of the shy, sensitive, introverted Malay is apt to terminate."

Again, in the more purely intellectual sphere, doubt is a state of conflict between beliefs. Doubt is not the opposite of belief, for this is unbelief and we know how, in a certain type of neurotic, all is doubt, even over the most obvious and to most people axiomatic truths.

What I wish to suggest in this paper is that the real nature of repression is as follows. Two complex patterns such as we have described in the beginning, in themselves antagonistic in the nature of the end they serve, fail to be integrated by the higher functions of the cortex or the mind—using the term 'cortex' if we are thinking physiologically, and 'mind' if we are thinking psychologically. Under these circumstances these patterns cancel each other out in the same way as do the patterns of muscular action, vegetative action and emotional action already discussed. Indeed, examples of each or all of

these minor pattern cancellations and oscillations may be found appearing as symptoms which modern psychology teaches us to be due to repression. These comprise pathological doubts and anxiety and the physical symptoms of angoisse. We thus get a view of repression as an interlocking of two opposing patterns which may be of equal value psychologically, and the outcome of such repression may be an eventual smooth integration or recovery ; or one side may more or less suddenly give way either partially, so that the opposing pattern becomes partially active in the form of a new symptom, such as an obsession lasting a comparatively long time or as a dream manifestation whose appearance is only momentary ; or wholly, in which case there is a sudden outburst comparable to McDougall's example of the Malay running amok. This occurs more often in the psychoses than in the psychoneuroses, though the 'all or none' reaction is familiar enough in the latter. In the ordinary conceptions of repression there is a tendency to represent it as if something superior was sitting on something inferior—as if, for few of us can avoid mental pictures, a virtuous child were sitting on the lid of a jack-in-the-box. But we do not always find the repressed material so much inferior, at any rate from the physiological point of view, and so I prefer the picture of two wrestlers locked in a clinch.

As an example of repression we may cite the case of a man who presented the following psychological situation. He was an only child and had in early life developed strong emotional attitudes towards his parents. He felt great inferiority towards his father more particularly in adolescence, when the latter had openly expressed intolerance at the son's clumsiness in manual activities demanding dexterity. Later the patient had entered upon a scientific career which had been crowned with considerable success, and had proved that he was definitely his father's superior in the intellectual field. The father was a man of narrow and somewhat bigoted religious views, and amongst other difficulties the son expressed great conflict in his religious life, being obsessed with the fear that if he took any rational attitude towards religious matters an offended and vengeful deity would exact some terrible retribution. Here it would seem that a religious complex in which his father's dominance and the emotional attitude towards this dominance was 'locked' with his scientific complex. Under ordinary circumstances this conflict is of course a common one, but it usually solves itself after a swing in either direction, in which the dogmatism of youth refuses credence either to religion or to science, by the adoption of a compromise such that a realization is possible that the two beliefs are not so incompatible as at first appeared. In spite of his intelligence and openmindedness in other matters, such a compromise was impossible for our patient, and the two patterns held the personality rigid in this respect, with frequent distressing oscillations at all levels of mental activity in the form of symbolic doubts, anxieties, and vegetative disturbances.

To continue our analogy referred to above : the wrestlers must turn towards each other, they must be interested in each other, they must find

contact all the time ; and when we translate this into terms of neuronic patterns I submit this represents repression. But suppose we find two individuals who never come into contact, who are never interested in each other, who continually turn their backs on each other. Translate this into terms of neuronic patterns and I submit we have the state of affairs in dissociation. Instead of antagonizing each other, each goes his own way, as perhaps happens at the levels of muscular action in cerebellar ataxia ; there is no junction at their synapses and so there is no interaction whatever.

This difference in reaction between neuronic patterns is probably fundamental, depending on the temperament of the individual. It is surely significant that, on the whole, repression tends to occur in the introvert and dissociation in the extravert. It must be remembered of course that no man is entirely introvert or extravert at all times and in all circumstances, and therefore just as introversion and extraversion are not mutually exclusive in any individual neither are repression and dissociation. However, the introvert, being concerned with his own inner subjective reactions, tends to get his patterns into conflict, antagonising each other as has been described. The extravert, on the other hand, is concerned with the object and his adjustment to it, and so, when integration fails, one pattern becomes adjusted to one object in the environment and another pattern to a second object. If then these objects are incompatible and the higher levels of cortical function fail, dissociation is bound to occur. This is best illustrated by the extreme case of the double personality in which major patterns are adjusted to incompatible life situations. In this connexion not only is the function of integration at fault but that of discrimination also fails, for presumably it is by this function that objects are discriminated and their suitability for synchronous adjustment determined. Until we can estimate what is the physiological difference between the introvert and the extravert we cannot go further in the explanation, but this may turn out to be chemical rather than structural.

To sum up, repression and dissociation are unsatisfactory methods of adjustment to conflicting situations. They are made necessary by the imperfection of the cortical functions of integration and discrimination. This imperfection may be due to a failure in complete development of the highest cortical levels in those individuals who are commonly described as always neurotic. On the other hand, the same imperfection may be permanently or temporarily induced by physical or mental trauma in an individual who under ordinary circumstances has these functions sufficiently developed to carry him through life. Here trauma is used in the widest possible sense to include every sort of injury from violence, from toxæmia, from fatigue, from emotional strains and every sort of environmental or endopsychic difficulty ; and physiologists must remember that these higher cortical functions being latest in development are most liable to interference and that they do not need a crow-bar to throw

them out of gear. Repression is an introvert syndrome, and may be represented physiologically by a cancellation of activities between two major patterns accompanied by more or less oscillation in the form of inco-ordinated symptoms characteristic of various levels of neuronic activity. Dissociation is an extravert syndrome, and may be represented physiologically by a complete failure of the passage of any activity from one major pattern which represents adjustment to an object to another major pattern representing adjustment to another incompatible object in the environment.

A NEUROLOGICAL TEST FOR STUTTERERS.*

By

ROBERT WEST, Wisconsin.

INTRODUCTION.

The psychological and medical worlds are now fairly generally aware of the theory that dysphemia† (stuttering) is often caused by an experience or series of experiences that produce an emotional set or feeling-pattern, usually (a feeling) of social inadequacy. The appearance of the symptom of incoördination of speech in situations in which this feeling of social inadequacy is most strongly elicited has been regarded as good evidence as to the cause of the disorder. Plenty of cases of marked and definite 'inferiority-complex,' however, show no stutter at all. Many cases, also, show dysphemia without any discoverable inferiority feelings, or any other emotional problems or disorders clearly distinguishing them from the non-stutterer. It seems, therefore, as though there were another factor in the etiology of dysphemia in addition to these psychogenic conditions—another factor that may or must combine with these emotional habits to produce the dysphemia. The present writer has been attempting to secure an objective measure of this unknown. He presents statistics on 64 cases, with the suggestion that we have, by means of the test here described, at least a distant view of the organic or structural factors in the stutterers' problem. The direction of his study was partly suggested by the fact that certain cases of dysphemia (or what at least closely resembles it) brought on by an organic lesion (luetic) and disappearing with the cure of the infection, showed very sluggish movement of the muscles of the face and mouth. These muscles were definitely sluggish in non-speech movements as well as in articulation.

TECHNIQUE.

Working with this clue in mind, we arranged a test of the maximum speed to which these facial and mouth muscles could be forced in purely repetitive acts of a simple nature. A Morse key was connected to an electro-magnetic recording lever, whose writing-point was in exact vertical alignment with that of a chronograph recording fifths of seconds, and both levers were caused to write upon a moderately slow kymograph. The subject was first instructed to close the key with his jaw, holding the apparatus in his mouth like a sandwich. He was given a little practice to enable him to learn the simple trick

* From the University of Wisconsin Speech Clinic.

† I use this term out of deference to the recent report of the Committee on Terminology of the American Society for the Study of Disorders of Speech. Dysphemia is also recommended by J. M. Fletcher in his recent work, "The Problem of Stuttering."

of closing the key firmly with each closure of his jaw. Then he was told to exert every effort to produce maximum speed. The speed on this trial was recorded.

The instrument was then mounted in such a way that he could conveniently rest his eyebrow on the edge of the rubber disc handle of the Morse key. By the drawing downward of his eyebrow, he could then close the key. After preliminary practice, he was told to make these movements as fast as possible. The speed on this was recorded. A combined rating was made by simply adding the number of jaw movements per second to the number of brow movements per second (see Tables I, II and III for the data). The subjects were all adults from the ages of 20 to 40. Most of them were under 25. With these adults there was no correlation between age and the speed of muscle movement.

RESULTS.

Several rather interesting facts appear :—

1. The 21 normal males showed a greater spread or variation than the 18 normal females.

2. The normal males were 1·11 units faster than the normal females.

3. The normal males were 1·76 units faster than the 18 stuttering males.

4. The 18 normal females were 2·17 units faster than the 7 stuttering females. The difference between the normal and the stuttering females is within ·41 units of the difference between the normal and the stuttering males.

We have as a check upon the reliability of these differences between the stutterers and the non-stutterers not only the low probable errors for these differences, but also the very significant parallel between the males and the females. The female stutterers not only differ from the female normals by about as much as the male stutterers differ from the male normals, but they differ *in the same direction*. The findings for one sex provide a convincing check upon the findings for the other, a check that is all the more impressive when we note that in the case of either sex the probable error of the difference is so low as to make the difference a matter of certainty.

To make the comparison in still another way : Note that the group of stuttering men overlaps that of normal men in 11 cases out of their combined total of 39 (see Table III). If there were a complete overlapping, we might safely say that our test is of no real significance ; if there were no overlapping, we might say that we were testing the only significant factor in the problem of dysphemia. The overlapping in our data, however, is completely confined to the lower quartile of the normals and the upper quartile of the stutterers ; the middle group of 10 cases among the stutterers and the middle group of 11 cases among the normals are mutually exclusive. Since the overlapping includes not more than a quarter of either group, we can at least say that the factor that we are testing is probably a more significant determiner of dysphemia than any other single condition. This contention is seen to be all the more

obvious when we note that, with respect to the overlapping in the case of the women, not a single stutterer falls within the range of even the middle 50 per cent. of the normal group.

On the basis of the combined evidence from the two groups, male and female, we may therefore assume that, after allowing for fundamental sex differences, sluggishness of response of certain facial and mouth muscles is either a cause of, or is related to a cause of, dysphemia.

Thus far we have drawn conclusions concerning our subjects as they may be arrayed in groups having certain demonstrable central tendencies. What can be said of the individual patient or of the individual normal subject with a low rating, about whose speech history we know nothing? (See Table III.) Patient No. 18 has a very low rating. Not a single normal male falls as low. We may fairly say of him that a certain quality or condition which marks the stutterer as different from the normals he possesses to a high degree. Patient No. 1, on the other hand, has a relatively high rating. Eleven normal males appear lower in the scale than he. We would say of him, therefore, that this certain quality or condition that characterizes dysphemia he possesses only to a slight degree. Other factors must be present to combine with this neuro-muscular sluggishness to produce the symptoms of dysphemia. It is not unreasonable to suppose that prominent among these other factors is poor mental hygiene. Suffice it to say at this point that the case history of patient No. 1 reveals a family background that most mental hygienists would regard as distinctly unwholesome for the maturing personality. Normal subject No. 21 has a rather low rating; only three male stutterers fall below him. We can only say of him, that, in spite of his low rating, the combined effect of all the factors predisposing to dysphemia is still not sufficient to produce it; and that, if thorough mental hygiene is necessary to prevent the average person from stuttering, his must have been doubly thorough.

DISCUSSION.

There are probably about 10 male stutterers per thousand in the normal population of America, or in other words the average male has one chance in 100 of being a stutterer. Whether or not this is an accurate ratio is not important here. What does concern us is how a given man's rating on this test relates itself to his normal chance of escaping this disorder. In our artificial sampling we have 39 males, 18 of whom are stutterers—a ratio of 462 cases of stuttering per thousand. From an analysis of Table III we see that, of the 10 male subjects rating 7 or below, eight are stutterers, or that their ratio is 800 per thousand. Of the 18 subjects rating 8 or below, 13 are stutterers, showing a ratio of 722 per thousand. Those rating 9 or below exhibit the ratio of 16 in 27, or 592 per thousand. Those rating 9 or above show a ratio of 1 in 13, or 77 per thousand. We say, therefore, that a subject rating 7 or below has almost twice the normal chance of being a stutterer; a subject rating 8 or below has about 1·5 times the normal chance of being a stutterer;

a subject rating 9 or below has 1·4 times the normal chance of being a stutterer: and the subject rating 9 or above has exactly one sixth of the normal chance of being a stutterer. The stutterers as a class differentiate themselves from the rest not so much by their low ratings as by their mediocrity. To have a low rating does not so much increase the subject's chances of stuttering as to have a high rating decreases it. To have a tendency to dysphemia may be normal, and not to have such a tendency may be the abnormal or supernormal condition. From that point of view our test, as applied to a given stutterer, would be more positive as a diagnostic index if it showed a moderately high rating than if it showed a moderately low one. The interpretation from the former case would be that the stuttering was probably not due to neuromuscular sluggishness but to other causes, while in the latter case one could be sure of nothing. A *very low* rating, of course, would be strongly presumptive of a neuromuscular etiology of the case of dysphemia under study; and a very high rating would probably never be obtained from a stutterer.

THE TAPPING TEST.

A great deal of work has been done with the tapping test, timing the rate of movements of the muscles of the arm and hand. It is generally agreed that men are, as a class, faster than women in this test. Most workers with this test seem to regard the rate as basic, dependent upon more or less fixed neurological conditions, and not to be increased by training. If this be true of the movements of the jaw and eyebrow also, we need not look to the training of the muscles of the jaw and face as a cure for dysphemia. We have been unable so far to demonstrate any increase in speed on training. In the "double-tongueing" of a cornet tone we have a simple repetitive movement analogous to the jaw movement. Cornetists state that a beginner at this movement attains a maximum speed at the start, although control, regularity, and prolongation are acquired by practice.

On this point Ream* in his work with hand-tapping states: "The extended practice experiment of the present study seemed to indicate that the test measures fundamental basic abilities, since improvement was on the whole conspicuously lacking. The inference is that the motor neural set, undoubtedly an important condition of such basic ability, is inherited."

What is this condition or quality that we are studying by means of our jaw-brow tapping test? Let us take as an analogue the simpler and better known hand-tapping test.

Clearly one of the factors limiting the speed of the repeated performances is that of the check or arrest of movement of one group of muscles and the starting of the movement by their antagonists. The speedy functioning of this reciprocal innervation depends partly upon the rapid routing through the cerebral cortex of the kinæsthetic impulses from the tendons and joints of the

* The Tapping Test, Psychological Monographs, Vol. XXXI. No. 1, 1922.

members involved in tapping. Cerebral inhibition in this case is but a matter of so routing the afferent impulses from any muscle group that the very act of contraction will furnish its own immediate inhibitory check, and also the excitatory impulse for contraction of antagonistic groups of muscles. The tapping test, therefore, is partly, it may be largely, a test of the efficiency of the cerebral mechanism for shifting inhibition. Evidently this efficiency increases during childhood, if one may judge from a few cases of children whom we tested. A more complete study of this development of tapping speed during childhood is given by Ream. He shows an increase for boys from less than 4 taps per second at the age of five to more than 6 taps per second at the age of 10, as over against about 9 per second for adult males. The maturing of the central nervous system, therefore, is accompanied by an increase in this inhibitory control.

The question now arises: why is it possible to develop so much more speed with movements of the hands and arms than with the masticatory and facial muscles? There are two possible answers: (1) That muscles of the arms and hands are supplied entirely with crossed nerve fibres. All their nerve connections are with the opposite side of the brain. The facial and masticatory groups, however, are supplied both with crossed and ipsilateral connections. Two sides of the brain are involved. In the complete cordination of both sides more synapses are involved. The passage through synapses involves time. This was partially brought out in our investigations by the finding that in alternate tapping of the right and left hands either hand moves about two-thirds as rapidly as when it alone is used in the tapping. This delay is probably a measure of the time involved in the synaptic connection between hemispheres in the process of cordination between the two sides. This rate of the right hand (in alternation with the left) is about that of the jaw. It is well known that the muscle groups having the greatest ipsilateral nerve supply are those of the upper part of the face. Accordingly we find these muscles slower in their rate of repeated contractions than those of the jaw. That, however, there are here involved other factors than the varying amount of ipsilateral nerve supply, is evident when we notice that many movements of the articulatory muscles are performable no faster than those of the upper face. We tried, for example a movement puckering or pursing the lips so as to extend them forward, while holding the jaw set and immobile; a movement retracting the angles of the jaw by means of the risorius muscle; a movement of the mandible from side to side; a movement of the palate opening and closing it against the emerging air-stream, while keeping the lips closed. These movements are slower than those of the masticatory movements and not demonstrably faster than those of the upper face. We must look, therefore, for another factor determining the speed of the reactions of these articulatory muscles, a factor that is clearly lacking in the set-up for the musculature of the hand and arm.

(2) A second guess, a very possible one, as to the situation that determines or limits the speed of these movements, is based upon the more direct and intimate connection of the musculature of the face, mouth, and throat with subcortical centres for the control of innate synergic vegetative and emotional reaction patterns. I refer to such centres as those for breathing, in the medulla; for coughing, sneezing, vomiting, yawning, gagging, and swallowing, partly mediated in the medulla (probably); and for crying, laughing, sobbing, frowning, smiling, etc., in the thalamus and striate bodies. These centres give off a profuse supply of upper neurones to the anterior horn cells of the lower motor fibres supplying the innervation for all of the musculature concerned in the processes enumerated above. These centres, on the other hand, have relatively few direct connections with the musculature of the arms and hands. It is probably this difference that is most highly a determining factor causing a difference in the speed with which inhibition may be shifted from one muscle to another. In the shift of inhibition from the extensor to flexor group of the arm, the only limiting factor (aside from the conduction time of the nerves and the latent period of the muscles) is the time involved in a reciprocating synaptic arc through one hemisphere of the cortex; while in the shift of inhibition from the risorius to the orbicularis oris muscle the limiting factors include all those involved in movements of the arm, plus the time involved in synaptic connections with the opposite hemisphere, plus the time involved in releasing the muscles concerned from the control exercised over them by subcortical centres. This process of releasing involves additional synaptic connections and is probably responsible for a large part of the difference in time between the movements of the face and those of the arm.

In short, our test may be largely a measure of ability to maintain an efficient cerebral control over the lower motor neurones to the face, mouth, and throat in opposition to the subcortical control of these same neurones. This explains why not a few postencephalitics and paretics stutter. This intermittent dysphemia may be regarded as an evidence of a comparatively even conflict between the cerebral and subcerebral centres for the control of the lower motor neurones. In rugged, sound speech that is seldom thrown out of rhythm, even by the greatest emotional upset, the cerebrum is clearly dominant; while in the spastic, scanning speech of the postencephalitic and of the bulbar paralytic the balance of power in the conflict for the control of the lower motor neurone is in subcerebral centres. Dysphemia is that condition of 'hair-trigger' balance between the two controls such that certain emotional conditions can intermittently shift the dominance from one to the other. This shift of dominance is characterized by the tonic and clonic spasms that we know as stuttering.

ETIOLOGY.

The difference between men and women in our test of the musculature of mouth and face is probably to be regarded as a rather fundamental sex difference. That it is not much related to speech is evident when we remember

that the incidence of dysphemia among men is much greater than among women. In comparing the two sexes from the point of view of speech we must conclude that the specific rate is not the significant factor in the disturbance of cerebral control but rather the difference between the individual's rating and the basic rate for his own sex.

With our 64 adults we found no correlation between age and speed of muscle movement. If this speed is linked up with the developmental history of the child, it is evident that it reaches a static point before maturity. The few children whom we have studied thus far show distinctly slower rates in our tests than adults. We are inclined for two reasons to assume tentatively that these cases have given us a safe lead as to the differences between adults and children in our test (this matter is now being verified). In the first place, with respect to sex differences, our results with the 'jaw-brow' test parallel the results obtained by various workers with the hand-tapping test; and, secondly, these investigators have also found children distinctly slower than adults. As in the case of women, we should not regard the specific rate as being a significant factor predisposing to dysphemia, but should refer the articulatory spasms to a disparity or lack of normal balance among the nervous centres concerned with the control of the speech organs, a thing measurable only by a comparison between the patient's tested rate and the normal for his age and sex.

Speaking generally as to etiology, we can say that two types of imbalance are responsible for this neuropathology predisposing the subject to dysphemia: (1) the interference with the inhibitory function of the cerebrum over lower centres through vice of one or both of the hemispheres or of their connection with the lower centres; (2) the abnormally heightened activity of subcortical centres causing their impulses to break through the cerebral inhibition. This hypertonic condition may be referable to toxic conditions systemic in nature, or to specific stimulation of the centres, as in chorea. Speaking generally as to the possibility of a remedy for this basic neuropathology predisposing to dysphemia, we can say that, if the disturbing condition is the result of disease, any exercises of the muscles of the face, mouth, and throat that increase the efficiency of their voluntary control are to be recommended. Just as, after many diseases paralysing arms or legs, judicious exercising will restore some degree of function by regenerating nerves and developing other tracts for those completely destroyed, so it is often possible to remedy pareses of the speech apparatus. Results of treatment in cases of this type lead us to make them the exception to the general rule that stutterers should not be given articulatory exercises. (It should be said, however, that these cases are few.) If, however, the predisposing factor is not a result of disease, but of inheritance of the brain structure and of the distribution pattern of the motor nerves, it seems quite likely that it is irremediable. In all probability exercises of the articulatory muscles will avail nothing.

SEX DIFFERENCES.

In the explanation of the comparatively greater incidence of dysphemia among men than among women (quoted by various authorities as from 4 to 1 to as low as 2 to 1) some importance must be attached to the greater variation of the normal males as against that of the normal females in the rate of jaw movement and in the rate of brow movement. In each of these tests of the functioning of facial and mouth muscles, the mean deviation for the males is approximately one and one-half times that of the females, while in the tapping test it is almost twice that of the females. Nature seems inclined to experiment more widely in the proportions in which the various elements are mixed in the male make-up. In this wider range of proportion the possibilities for imbalance among the brain centres is increased. To set up a statistical analogue: arrange 50 cards numbered 21 to 70 (series A); arrange 50 other cards bearing even numbers from 2 to 100 (series B). Series B, having the greater range and the higher average represents the variety of coördinational elements out of which nature selects a combination for each male; series A represents the range of nature's choice for the component elements of the female. At random choose two cards from series B and two from series A. This is as though nature were constructing first a boy and then a girl. The two cards from series B may be 98 points apart. The two cards from series A cannot be farther than 49 points apart. Suppose we assume arbitrarily that the point at which dysphemia will appear in either sex is when the difference between the two cards drawn is 40 or more. In series B 465 possible combinations will produce a difference of 40 or more; in series A there are only 55 such combinations. In each series there are 1,225 possible two-card combinations.

If these combinations are selected by chance, and the cards returned to the pack after each drawing, 10,000 drawings from series B, determining the inheritance of boys, will, in our simple analogue, produce 3,800 stutterers; while 10,000 drawings from series A, determining the inheritance of girls, will yield only 450 stutterers. On some such disparity of combination-possibilities the greater incidence of stuttering among men may depend. Of course the combination is probably not of two variables, but of many.

Many writers have attempted to explain the greater incidence of dysphemia among boys on the basis of the difference of social demands made upon the two sexes. The sexes may be regarded as having different social environments, and the environment in which the boy is placed is less hygienic. Perhaps in answering the question as to why either sex stutters at all, we should in the future emphasize more than we have in the past these predisposing neurological factors. These conditions render the speech co-ordination so liable to interruption by muscle spasm that whenever the social situation is even slightly upsetting the patient begins to stutter.

CONCLUSIONS.

On the basis of our data, pending further investigations, the following conclusions seem warranted:

(1) Some stutterers present such a serious neurological problem that they should not hope for a 'cure.' To attempt to remedy such a case of dysphemia by probing for some psychic trauma is not only futile but likely to produce in the patient a morbid introspection.

(2) Some stutterers offer not neurological but psychological problems and should react favourably to mental medicine.

(3) The adequate handling of any case of dysphemia should involve a differential diagnosis separating the neurological problem from the psychological.

TABLE I.

RATING OF SIXTY-FOUR SUBJECTS.

(1) Combined rate of movement of the jaw and brow.
(2) Hand-tapping rate.

Number of cases.	1		2	
	Means	Probable Error of means	Means	Probable Error of means
21 males, normal	9·26	·262	7·98	·159
18 males, stuttering ...	7·50	·172	6·93	·126
18 females, normal ...	8·15	·148	7·21	·88
7 females, stuttering ...	5·98	·295	7·53	·258

NOTE.—The hand-tapping rate was taken to check our technique by comparison with other studies.

The probable error was found by: P.E. of M. $= \frac{\cdot 8453 \text{ M.V.}}{\sqrt{N}}$

TABLE II.

DIFFERENCES IN THE JAW-BROW TEST.

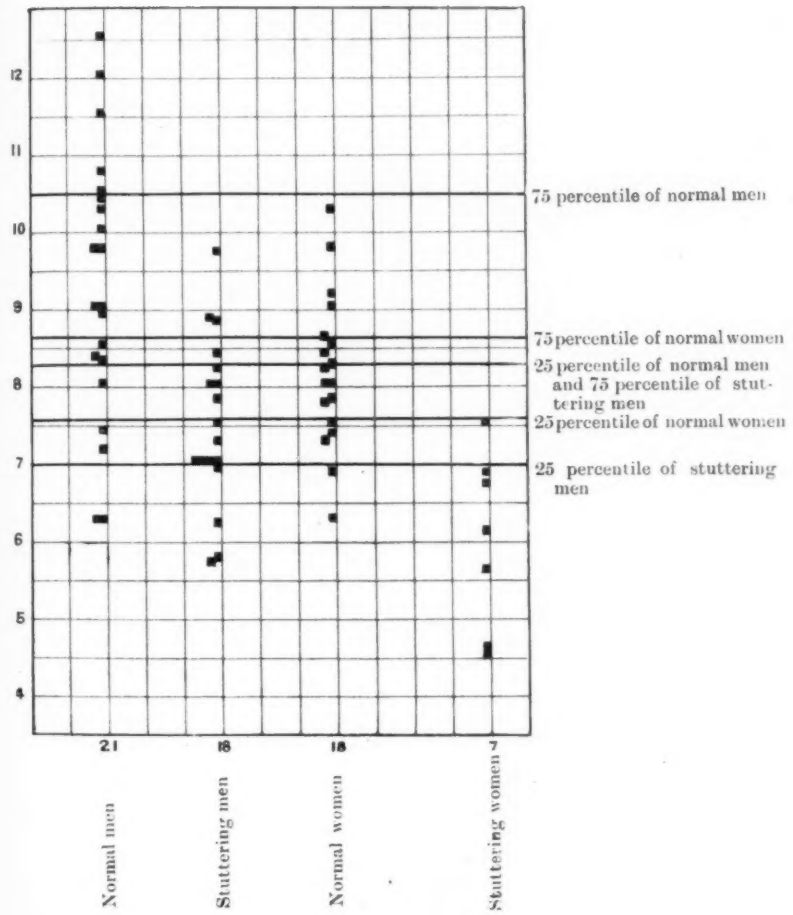
Between	Difference	Probable error of difference	D/P.E.	Probability of a real difference
Normal and stuttering males ...	1·76	·313	5·62	9999·2 in 10000
Normal and stuttering females	2·17	·33	6·58	10000 in 10000
Normal males and normal females	1·11	·30	3·70	9937 in 10000
Stuttering males and stuttering females	1·52	·341	4·45	9987 in 10000

NOTE.—The P.E. was calculated by: P.E. of D. $= \sqrt{\text{P.E.}^2 \text{ M}_1 + \text{P.E.}^2 \text{ M}_2}$.

The probabilities were derived from Table 45 on page 200 of "Mental and Social Measurements," by Thorndike.

TABLE III.

DISTRIBUTION OF THE SIXTY-FOUR SUBJECTS.



Critical Review.

THE NATURE AND SIGNIFICANCE OF SENILE PLAQUES.*

BY

MACDONALD CRITCHLEY, LONDON.

FEW problems in neuropathology have aroused more discussion than the question of origin and development of so-called miliary or senile plaques. Although more than thirty years have passed since the first adequate description, the histological nature of these bodies is still a matter of controversy. An even greater uncertainty surrounds the exact clinical significance of the plaques, and their importance in the pathology of the senile psychoses.

The first reference to the existence of senile plaques is contained in a paper by Blocq and Marinesco in 1892, on the pathology of epilepsy. In the brain of an aged epileptic they found small circular masses, measuring about 60 μ in diameter, scattered throughout the cortex. These bodies were conspicuous on account of their deeper colouration. A vague stippling of the interior suggested to the authors the possibility of their being "nodules of neuroglial sclerosis."

There appeared in 1898 the first important record of these nodular masses in a paper by Redlich who described them in the brains of two senile epileptics. Redlich spoke of them as "miliary scleroses." Other contributions followed, notably those of Perusini, Alzheimer, Oppenheim and others. Miyake described them as "glial rosettes," Léri as "sclérose névroglique miliaire." The term "senile plaque" was first proposed by Simchowicz. Fischer's contributions in 1907, 1908 and 1910 were the next important landmark in the study of the plaques; this author proposed the word "Drusen" or "drusigen Nekrosen," and also—from their resemblance to the lesions of actinomycosis—"Sphärotrichien." Later writers have usually employed the term "senile" or "miliary plaques," though in Germany one still speaks of "Drusen," "sphærotrichea cerebri multiplex" or "Redlich-Fischer plaques."

More recently, valuable contributions to the problem have been made by Rizzo and Bolsi in Italy, Uyematsu in the United States, Marinesco in Roumania, Hechst in Hungary, Divry in Belgium and Bouman in Holland.

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HISTOGENESIS.

In the broadest terms, senile plaques may be described as small, circular areas of densification, scattered throughout the cerebral cortex. They stain intensely with silver-impregnation methods, such as those of Bielschowsky or Levaditi. Special tinctorial methods, as those of Hortega, demonstrate the presence of microglial elements, while other stains, such as Cajal's silver method, may reveal neurofibrillary structures. Histochemical tests may also give evidence of amorphous lardaceous bodies or aggregations of lipoid.

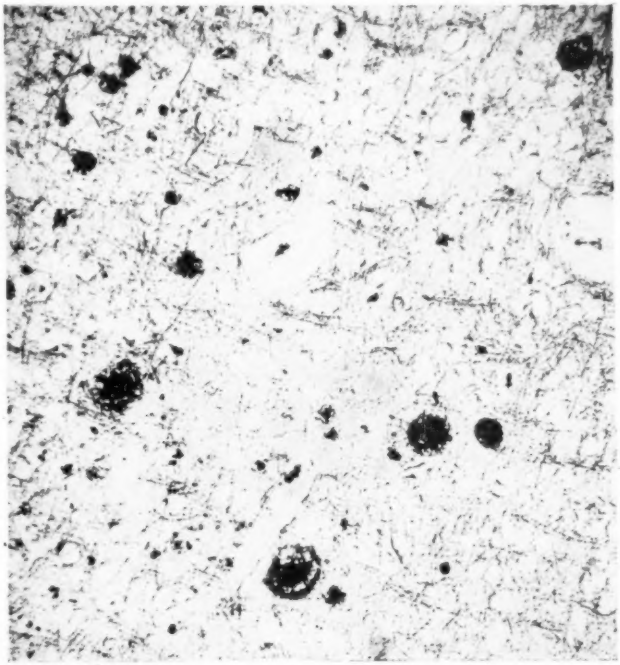

FIG. 1. Cortex of a senile dement, aged 91, showing numerous plaques of various sizes. (Levaditi stain).

Minute study of the plaques demonstrates a remarkable variation in the finer structure, but numerous *form-constants* can be recognised, including rings, radial figures, star-shaped bodies, and circular homogeneous areas. It is doubtful whether these different forms represent distinct types or merely transition stages.

Following with some modification, the classifications of Uyematsu, Marinesco and others, one may speak of three main varieties :—

1. A diffuse or homogeneous form.

2. A spheroidal or ring-like form containing a central cellular body.
3. A spheroidal or ring-like form containing in the centre an amorphous non-cellular body.

Uyematsu also adds to these a perivascular variety.

1. The first type is made up of circular areas measuring from 10μ to 150μ in diameter. Their structure consists of a mass of fine, interlacing fibrils, entangled together to form a meshwork. At times, a few nuclei are

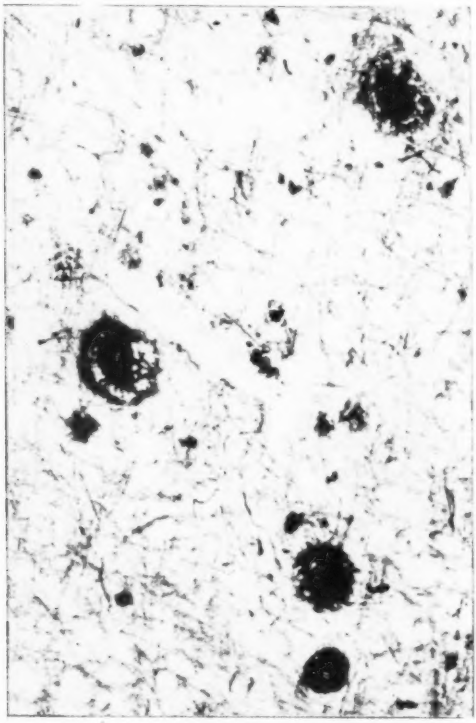

FIG. 2. High-power representation of same specimen as Fig. 1.

visible in the interstices. The fibrils are strongly argentophil in their staining reactions. In some of the plaques of this type the interior is relatively less dense than the peripheral part.

2. In the second variety, one recognises an outer ring of variable thickness, made up of a fibrillary network with occasional cells. Alzheimer called this zone the "Hof" in contradistinction to the central "Kern." Sometimes cells lying immediately outside the plaque send in fine processes which pierce

the peripheral ring. Bounded by the outer ring is a narrow, clear zone surrounding a central, nuclear-like mass. In this particular type of plaque, this consists of a cellular body with a circular or oval light-staining nucleus. This structure is also argentophil but varies in its power of taking up stains. Thus, in some cases, the external ring stains deeper than the interior, while in others the reverse is true. With appropriate technique, granules of lipoid may be seen within the cytoplasm. In a number of instances the fibrils of the peripheral area tend to assume a radial arrangement, constituting the so-called 'morning-star' appearance.

3. The third type resembles the second except for the character of the central mass, which is amorphous, acellular, and usually of irregular outline.

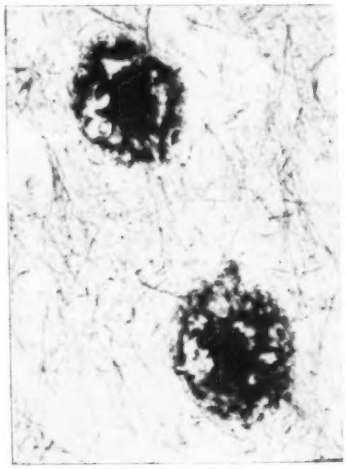

FIG. 3. Still higher magnification, showing the detailed structure of three plaques (x 500).

This portion differs from the periphery in its lack of affinity for silver stains. Divry has demonstrated the bi-refractile property of these bodies.

More detailed histochemical study of the plaques, particularly since the use of the Hortega methods, has brought to light several important points in the structure :—

(*a*) The plaque as a whole is essentially argentophil.

(*b*) There may be a very narrow acellular zone surrounding the periphery of the plaque (=the " zona x " of Tumbelaka).

(*c*) The acellular central masses have the histochemical and physical properties of amyloid or of some analogous substance (Divry *et al.*).

(*d*) The plaques do not react to neuroglia stains although in the older varieties large neuroglial spider-cells may be seen in the vicinity.

(*e*) Appropriate staining methods reveal the presence of lipoid within the central portion as well as inside the fibrillary zone of the plaque.

(*f*) Hortega methods reveal an important participation of microglia cells. In the plaques belonging to the first type they may be entirely absent, but are particularly frequent in the other forms. Microglia cells occur both in the peripheral and in the central zones. In the former case they may become swollen and their processes may hypertrophy. Within the innermost portion one or two microglia cells may be visible. Their processes spread out in a radial fashion and tend to swell as they traverse the peripheral zone. At a later stage, they undergo regressive changes: they distend, lose their processes and become amoeboid. Intracellular granules of lipoid material may be visible.

(*g*) Oligodendroglia apparently does not participate in the structure of the plaques.

(*h*) In the older plaques neurofibrils may be demonstrable by Cajal's silver stain ("neurotisation"). Occasionally, myelinated axis-cylinders may be seen.

Although Fischer and Oppenheim both emphasized the frequent close relationship of the plaques to the blood-vessels and capillaries, it seems that such an association is far from constant. Indeed, the majority of plaques appear to have no intimate connection with the vessels. On the other hand, a proportion of the plaques are obviously intimately related to and seem actually to grow in the wall of a vessel, which may be either healthy or diseased. Uyematsu, indeed, describes a fourth or perivascular variety of plaque. Bouman has given an interesting study of the development of this type of plaque from its earliest stages. According to his account the following processes may be noted (see illustration):—

(*a*) A bean-shaped homogeneous clump, of argentophil material, attached to one side of a blood vessel.

(*b*) The mass now surrounds the vessel in an asymmetrical fashion (the 'signet-ring'). The edge farthest away from the vessel wall stains darkest with silver.

(*c*) The outer wall of the clump now shows a ray-like formation resembling a brush.

(*d*) The rays become thicker and stain more intensely.

(*e*) The rays increase still further and become irregular. They grow out to a peripheral limiting horny ring in a radial or spoke-like manner (=the 'morning-star').

(*f*) The blood-vessel atrophies and the central clump shrinks.

Uncertainty exists as to the significance of the various types of plaque. On the one hand, they may represent entirely distinct varieties, differing essentially in histological structure. Alternatively, they may represent different stages in the development of an adult form; thus, type 1 might be regarded

as a relatively younger type, although Urechia and Elekes consider them as the older varieties. There is, however, a third possible explanation of the different appearances; the plaques may be regarded as spheroidal masses consisting of an external shell of fibrillary meshwork or felting, surrounding a core of a cellular or hyaline nature. Transection through the equator of such spherical masses will show them as annular areas with a central nuclear mass. Sections which do not pass through the diameter but merely 'top' the plaque appear as circular areas of homogeneous fibrillary structure, with or without a relatively less dense interior.

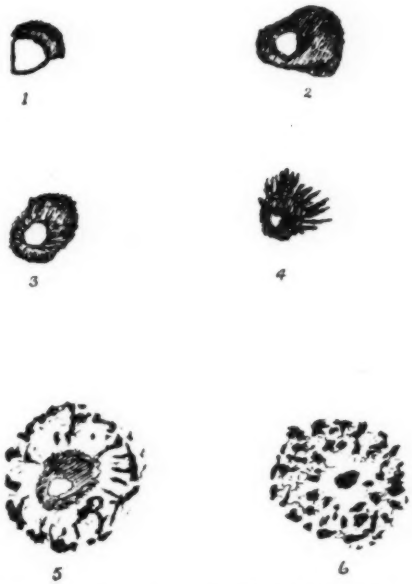

FIG. 4. Schematic representation of the development of the perivascular type of plaque. 1—6. (see text). 2: "the signet-ring." 5: "the morning-star." (After Bouman).

TOPOGRAPHY.

Senile plaques are practically confined to the cerebral cortex. Here, the frontal lobe, the cornu Ammonis, and the island of Reil show the maximum change. Plaques have also been described in the cerebellar cortex and in the medulla; they are never seen in the spinal cord. In the great majority of cases no plaques are to be found in the basal ganglia; Bouman and Bok, however, have recorded nine cases in which the putamen, claustrum and caudate were markedly infiltrated by plaques. They point out that their technique of

fixation may be of importance, and that since the routine employment of their new method plaques have been discovered in the basal ganglia "almost regularly." The general experience, however, is to confirm the rarity and diminutive size of plaques within the corpus striatum. It is perhaps worth emphasizing that in Bielschowsky preparations small bundles of nerve fibres, cut transversely, may give an appearance which superficially resembles that of the homogeneous variety of plaque.

Within the cerebral cortex the plaques appear chiefly in the deeper (pyramidal cell) layers. When very numerous the more superficial laminæ as well as the immediately subjacent white matter may contain a few; in such cases the larger plaques are situated in the deeper strata. When the cerebellum is the seat of plaque formation the molecular layer is the most usual site, although at times plaques may be seen near the Purkinje cell laminæ.

OTHER ASSOCIATED PATHOLOGICAL APPEARANCES.

The intrinsic nature of the anatomical circumstances under which senile plaques are found makes it probable that other pathological changes will coexist. Chief among the associated lesions are those which belong to the pathology of the senium. These include neuronal atrophy, neuroglial changes of both proliferative and regressive type, and intracellular pigmentary depositions. Of special importance are the intracellular fibrillary changes of Alzheimer; this lesion particularly often coexists with senile plaques (Bielschowsky, Uyematsu, Stief, Bolsi), although in Fuller's cases Alzheimer's fibrils were rare.

Lesions of an arteriosclerotic nature may be present in brains rich in senile plaques (Fuller, 62 per cent.; Uyematsu, 27 per cent.). There is a general agreement, however, with Fuller's statement that arteriosclerosis alone has but little direct causative relationship with the formation of plaques. Bleuler, indeed, stated that arteriosclerosis seems to give a certain amount of immunity against the development of senile plaques.

Sigg drew attention to the constancy with which sclerosis of the basal arteries occurred in his series of plaque-containing brains, and he tentatively regarded his cases as lying midway between senile brains with soft blood vessels and those of arteriosclerosis with intensely hard arteries.

In his study of the glia in chronic vascular disease of the brain Globus has noted circumscribed, rosette-like aggregations of microglia. Such bodies need to be distinguished from senile plaques in histological studies.

Laignel-Lavastine and Tinel have described in the cortex of senile brains irregular "fern-leaf" precipitates of argentophil material. These deposits are larger than the ordinary senile plaques and produce no reactionary change in the nerve cells of neuroglia. They are only exceptionally associated with the ordinary senile plaques. On account of their physical properties and staining reactions Tinel concluded that they were fatty in nature. The authors regarded these bodies as representing a variety of miliary plaque, of more rapid development; they were never found except in brains of senile dements and usually

in those whose clinical history had been short. Tinel looked upon the classical senile plaque as a "vestigial, cicatricial, and dystrophic relic" left by the re-absorption of his type of fatty plaque. In support of this view, he demonstrated in three cases structures which he regarded as transition-forms between the fatty or acute plaque and the more chronic senile plaque of Redlich and Fischer. Rizzo, however, believes that the fatty plaques of Laignel-Lavastine and Fischer are artefacts due to the methods of fixation, and he denies the existence of transition types.

THE NATURE OF SENILE PLAQUES.

Fischer's original hypothesis regarded plaques as the expression of a specific infection due to a streptothrix-like micro-organism. This view, however, never gained popularity.

Prior to the employment of the Hortega methods in the study of miliary plaques much uncertainty existed as to their exact nature. Some regarded them as originating from decayed nerve-cells (Redlich, Bonfiglio, Perusini, Bickel) with or without secondary neuroglial infiltration. It was pointed out, however, that the occasional finding of plaques in the molecular layers of the cortex and in the white matter is evidence against their neuronic origin.

Theories as to the primary glial nature of the plaques were held by Miyake, Léri, Ziehen and others, although it was found that the plaques fail to react to pure glial stains.

The researches of Ley and Timmer demonstrated for the first time the importance of microglia in plaque formation. Both healthy and pathological Hortega cells in the peripheral and central zones have already been mentioned as occurring in a large proportion, though not all, of the plaques. It has furthermore been shown that oligodendroglia does not participate in the structure of the senile plaques.

Lastly, the histochemical work of Divry showed that according to the optical properties, the staining reactions and the solubility in various solvents, the plaques are composed of amyloid, and he regards them as the expression of a disseminated hyalo-amylosis affecting the cortex.

The modern conception of the plaque is that of a reactionary change directed against a specific metabolic process of toxic nature. The probable successive steps in the development may be stated as follows: there is primarily an involutional alteration in the chemistry of the fundamental ground-substance wherein the elements of the nervous tissue live. (Both Timmer and Ley regarded the original metabolic change as being disintegrated microglia). In this way lipoid (or possibly amyloid) substances are precipitated which are strongly argentophil. These bodies provoke a microglial reaction: Hortega cells become incorporated and insinuate themselves into the innermost part: here they themselves undergo degeneration. Marinesco believes that necrosis of the microglia is the cause of the central depositions of amyloid material:

in this way plaques of type 3 would be of later development than those of type 2. Microglial cells also appear in the periphery ; later they hypertrophy and then break down and finally disintegrate.

Although macroglia plays no essential part in the histogenesis, at a later stage hypertrophied astrocytes may be seen collected in the vicinity so as to form a ring round the plaque.

Nerve cells and fibres may also become involved during the final stages ; according to Marinesco there may be an actual new-formation of nerve fibres, some of which attempt to penetrate the plaque. Here, however, the environment is unsuitable and the fibre-growth becomes arrested.

Why the cerebral cortex should almost exclusively be the seat of plaque formation has never been satisfactorily explained. Löwenberg suggested two possible hypotheses ; first, there might be some peculiarity in the cortical reticulum favouring plaque-formation. On the other hand the predilection might depend upon the presence of capillary fibrosis in the senile cortex.

THE CLINICAL SIGNIFICANCE OF MILIARY PLAQUES.

Most interest, and at the same time the greatest controversy, centre round the possible clinical significance of the senile plaques. As their name implies, they are chiefly encountered in the brains of the aged and particularly in cases of senile psychosis. The main problems are, therefore, whether plaque-formation is essentially an involutional process, or whether it is associated more intimately with psychotic states. Lastly, the possible specificity of the plaques in any particular clinical type of mental or physical change needs consideration.

It was believed by the earliest investigators that there might be a causal relationship between the presence of plaques and the clinical expression of epilepsy (either senile or idiopathic). Thus it will be recalled that the original cases examined by Blocq and Marinesco and by Redlich were of aged epileptics. Léri, in his thesis on the senile brain, refers to the occasional presence of plaques (miliary sclerosis) which, as he says," would seem to be associated with attacks of senile epilepsy, or possibly with dementia." In Fischer's series, fits occurred in five out of the 58 cases in which plaques were demonstrable ; in his control series of 53 plaque-negative brains, epilepsy occurred four times.

Later work clearly indicated that there was little or no direct association between plaques and epileptic attacks, and interest centred upon their occurrence in the senile or presenile psychoses. Thus, in 1906, Alzheimer referred to their presence in the malady which now bears his name, and three years later Perusini described three similar cases. The contributions of Fischer, however, appearing over the years 1907–1910, drew attention to the occurrence of plaques in cases diagnosed clinically as senile dementia, and in particular to their presence in that variety known as presbyophrenia. Thus out of his series of 110 brains of patients dying with various psychoses, plaques were found in 56 : of these 42 had been diagnosed as presbyophrenics, while the

other 14 were patients with paranoid, catatonic or manic-depressive states. In a later contribution Fischer expressed the opinion that the plaque-positive cases represented a definite clinical entity which he termed " presbyophrenic dementia " ; in cases of simple senile dementia no plaques occurred.

Oppenheim (1909) demonstrated plaques in six out of 11 senile brains ; of these, six were from patients with senile dementia with presbyophrenic trends and three were from cases of dementia senilis simplex with arteriosclerosis. Sigg, too, associated plaque-containing brains with the clinical picture of agitated states, and plaque-free cases with torpidity.

In the same year, Schönfeld demonstrated plaques in 22 brains from patients older than 50 ; the maximum incidence was in the ninth decade. The clinical diagnoses in the 22 cases were as follows : presbyophrenia 8 ; presbyophrenic trends 5 ; dementia senilis simplex 2 ; chorea with psychosis 2 ; undiagnosed cases 3 ; no mental change 2. Schönfeld concluded that plaques were constantly present in cases of presbyophrenia but that they were not pathognomonic of that condition.

Latterly, the correlation between senile plaques and presbyophrenia has been less insisted upon, and there has been a greater tendency to associate them with simple senile dementia or even the normal senium. Thus Uyematsu criticizes Fischer's conception of presbyophrenia as being unlike Wernicke's original description, and resembling rather the definition of ordinary senile dementia as formulated by the American Psychological Association.

How far one can speak of a ' normal ' or ' healthy ' old age is, of course, debatable, but it is noteworthy that plaques have often been demonstrated in brains of senile patients who had shown no obvious mental symptoms. Thus Fischer found plaques in two out of 35 brains of normal aged subjects ; Oppenheim found them in two cases of healthy old age. In the brain of a normal old man who died at the age of 105, Costantini found plaques, but only in very small number. I have had the opportunity of studying the brains of two aged subjects aged 99 and 102 respectively. In both cases only a very few scattered plaques were found, particularly in the precentral gyri.

Simchowicz's contributions are of particular interest ; he regards plaques as typical of the normal senium but believes that they increase with advancing years. He points out that it is not sufficient merely to record the presence of plaques, but that their number, size and arrangement must also be considered. He therefore suggested that a so-called ' senile index ' should be taken ; the maximum number of plaques in a microscopic field (Leitz obj. 3, eyepiece 3, magnification 80) is noted. Examinations are made of different cortical regions, taking sections cut at a thickness of 20 μ, and stained by the methods of Bielschowsky or Mann-Alzheimer. The actual number of plaques in each piece is taken as a ' senile index ' and tabulated as follows :—

Index of frontal lobe – – – – I.F.
,, temporal lobe – – – I.T.

Index of cornu Ammonis – – – I.A.
,, parietal lobe – – – – I.P.
,, motor cortex – – – – I.M.
,, occipital lobe – – – – I.Occ.

The 'senile formula' is then recorded thus :—

I.F. : I.A. : I.T. : I.P. : I.M. : I.Occ. = 52 : 26 : 26 : 17 : 8 : 5
(in a case of senile dementia).

In a normal old man of 104, the formula was :—

I.F. : I.A. : I.T. : I.M. : I.Occ. = 8 : 5 : 3 : 0 : 0.

In Alzheimer's disease, the maximum plaque incidence was found to be in the visual cortex, thus :—

I.F. : I.A. : I.T. : I.P. : I.M. : I.Occ = 45 : 42 : 44 : 62 : 30 : 74.

The ratio I.F./I.Occ. is, therefore, important, in Simchowicz's opinion, in differentiating Alzheimer's psychosis from senile dementia on pathological grounds. According to this author's results, the differences between healthy old age and senile dementia are purely quantitative, and depend entirely on the actual number of plaques.

In cases of Alzheimer's disease, senile plaques are present in the cortex, but always in conjunction with the characteristic intracellular fibrillary alterations.

The present conception of the significance of the senile plaques is to regard their presence in large numbers as indicative of senile dementia, though not pathognomonic. As Fuller says, plaques occur with greater frequency in senile dementia than in any other form of insanity. Uyematsu's conclusions are particularly worthy of quotation : "All cases of senile dementia should show miliary plaques, and in those cases failing to do so ; a diagnosis of senile dementia is doubtful. . . . Miliary plaques do not necessarily indicate the diagnosis of senile dementia although their presence proves to be highly suggestive. By the absence of plaques a diagnosis of senile dementia can be ruled out. . . . The miliary plaques are characteristic of senile dementia and not for any special form of the disease." Much further clinico-pathological research is obviously necessary before Uyematsu's last statement can be accepted unhesitatingly. Thus it will be necessary to investigate more fully the clinical aspect of the plaque-free cases of senile dementia ; to enquire whether the size, number or arrangement of the plaques can be correlated with any distinct clinical variety of dementia senilis. In this connection the question of senile plaques in the corpus striatum would repay further investigation ; indeed there is some evidence to suggest that the clinical course of such cases may be outside the usual picture. Stief indeed has associated the presence of miliary plaques in the striopallidum with the picture of senile rigidity (the *senile Muskelstarre* or *Vorsteifung* of Jakob).

More study is also due to those rare cases in which senile plaques have been encounted in the brains of relatively younger individuals. Thus plaques

were present in the cortex of one of Simchowicz's patients dying from Korsakow's syndrome at the age of 71. One of Marinesco's patients, a female of 45, also showed clinically a polyneuritis with Korsakow's psychosis. A patient of Tiffany's dying at the age of 31 from tabes without mental change also showed numerous miliary plaques.

It is noteworthy that miliary plaques are characteristically absent from one very important variety of senile psychosis, viz., circumscribed cortical atrophy or Pick-Spielmeyer's disease. With the exception of the examples recorded by Kufs and by Altmann, and the fourth and distinctly atypical case of v. Braunmuhl, no instance is on record of the presence of senile plaques in Pick's disease, and in the cases reported by the two first mentioned observers the plaques were extremely small and infrequent.

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Editorial.

NEUROSYPHILITIC UNCERTAINTIES.

IT is probably the case that neurosyphilis is the outstanding disease of all those which have been associated with re-organised conceptions in the course of the last twenty or thirty years. A centuries-long era of mystery and empiricism came to an end with the discovery of the protozoal nature of the infection causing syphilis, the inevitable corollary being revision and re-systematisation of current views of its effects on the neuraxis and of the varieties of disease thereby produced. This wonderful spurt in the long race to the goal of perfected knowledge has been succeeded, however, by a period of 'marking time,' for the research that culminated in detection of the spirochæta pallida has started almost as many problems as it has solved.

The spirochæte discovered by Schaudinn and Hoffmann fulfils all Koch's postulates; its morphology is thoroughly determined; it can be cultivated outside the body and kept going for many generations. But under ordinary conditions it seems to excite disease in man only, and to have no intermediary host. Whether it has a life-cycle in any way comparable to that of other protozoa remains at present indeterminate; in the primary sore and in the last stage of general paralysis its form is identical, as is also the case through many successions of artificial culture. Various modified shapes have been seen, but whether these are in process of degeneration or represent a resistive adaptation is not known.

When the spirochæte passes by perivascular lymphatics to become colonised at various points of the cerebrospinal vascular system local tissue-reactions ensue. It is doubtless possible for the organism to make its way from Virchow-Robin spaces into the interstices of the parenchyma, especially if the mesothelium is weakened by toxic influence, but in this particular variety of neurosyphilis it is mainly the mesodermal elements which react. The attack on the nervous system by invasion of the spinal fluid produces histological effects of a different character. Until certain questions connected with the functions and

interrelation of subarachnoid fluid-system and cerebral blood-system are satisfactorily elucidated a description of the exact way in which the brain parenchyma is reached must remain to some extent conjectural. Experimental, progressive, neurosyphilis has never as yet been characteristically produced. It is known that perineural lymphatics are open to injection from the subarachnoid space, so that invasion of the fluid therefrom can be accepted as a possibility; but whether perineuronic spaces are infiltrated from perivascular lymphatics or subarachnoid system is undetermined, and the experimental evidence is conflicting. On the whole, it is not, so far, proved that the fluid is the nutrient lymph of the brain, or that normally there is a flow from one system to the other via parenchymatous spaces. Nor must the fallacy of arguing from normal to diseased conditions be lost sight of. But this may be said, that if the spirochæte penetrates the brain from the vascular side it is difficult to see why the occurrence is not far more common; if from the meningeal and fluid side, its action in this respect is very different from that of many other organisms which, heavily infecting the fluid and meninges, are nevertheless scarcely ever found in the brain itself.

The formerly much discussed problem whether pathological distinctions of import can be drawn between vascular and parenchymatous neurosyphilis has now become largely meaningless. Wassermann reactions are positive in both types; the spirochæte is found with ease in many cases once classed as parasyphilis; a large part of the lesions of general paralysis is meningovascular; 'primary' optic atrophy is, in point of histological fact, also meningovascular; experimental pathology has succeeded in producing gummatous development in the testis of the rabbit by inoculation with material derived from the cortex of the paretic. It is no exaggeration to say that general paralysis is 'more syphilitic' than the varieties formerly classed as tertiary syphilis. Nor can clinical distinctions be maintained with the same absoluteness as in other days; in particular, the time-factor once confidently relied on is of no differentiating usefulness. Pathological evidence shows neurosyphilis to be one and indivisible. The existence of widely differing clinical types must of course be acknowledged, though on occasion they run into each other; their development depends largely on paths and sites of infection and on variation in local reaction. Old distinctions to some extent continue, but are oriented from a new standpoint. The question of why one

route should be taken rather than another is at present quite unsolved, and scarcely less obscure is the collateral problem of why, after latency sometimes measurable in decades, nervous symptoms should arise. It is possible the clue is to be sought in the theory of sensitisation by toxins, tissue resistance being gradually undermined by a process of 'hyperallergy,' so that in the end a minute toxic dose produces an exaggerated response.

Of several unsettled questions perhaps the most difficult relates to the problem of where, and in what form, the organism lies dormant until awakened into activity by alterations in immunity or nutrition, or, conceivably, by trauma. The interval between infection and the development of clinical manifestations is sometimes almost incredibly protracted. In some conjugal cases the presumption is that the seminal fluid is the carrier (the spirochæte is larger than the head of a spermatozoon); but what process of 'hibernation' the organism undergoes for these long periods is quite unknown.

A further undetermined question concerns the admittedly great disproportion between the incidence of syphilis and the appearance of the nervous variety—according to a commonly accepted view the latter is a sequel in only some three to five per cent. of infected subjects. Certain lines of evidence, both clinical and experimental, appear to lend support to a hypothesis guessed at by Fournier and in recent years often regarded with favour, to the effect that neurosyphilis is caused by a virus of neurotropic properties, a spirochætal organism distinct from that responsible for syphilis with pronounced cutaneous manifestations, which is attributed to a dermotropic variety. Among the arguments for this contention are the observed facts that "mild syphilis is nervous syphilis" (Oskar Fischer), and that in numerous cases of juvenile tabes and general paralysis no cutaneous or mucous involvement has been seen. Nonne, too, has collected data showing that in the descendants of neurosyphilitic parents latency of primary and secondary symptoms is the rule. Reliance is often placed, further, on the experimental evidence of Levaditi and A. Marie, who obtained a dermotropic strain of organism from a testicular chancre in the guineapig (inoculated with human chancrous material), and a neurotropic strain from a similar guineapig chancre caused by infection with blood from a general paralytic; and who claimed to have demonstrated certain technical (cultural and other) differences between the two. But many serious objections may be raised to these lines of argument. Signs of

somatic syphilis are very common in cases of general paralysis (fibrosis of spleen, kidneys, liver, cerebral arteries); in that form of neurosyphilis syphilitic aortitis is often seen, exactly the same as that met with in somatic syphilis; the experiments mentioned above are vitiated by failure to exclude the possibility of the human chancrous material (from which the dermotropic virus was derived) itself on occasion containing the neurotropic kind. Most significant of all is the invasion of the meninges and the presence of fluid reactions at the stage of ordinary cutaneous and mucosal secondary rashes. The argument based on the rarity of neurosyphilis in certain less civilised countries where syphilis is rampant is unimpressive, the facts being capable of another interpretation. Besides, cases are on record of the development of parenchymatous neurosyphilis in Europeans infected from such semicivilised sources. On the whole, firm proof of the dualist theory is still to seek.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[71] **Nervous control of the electrical resistance of the skin.**—Curt P. Richter. *Bull. Johns Hopkins Hosp.*, 1929, xlv, 56.

In monkeys section of all nerves to the foot produces an enormous increase in the skin resistance of the plantar areas, containing many sweat glands, but has no effect on that of the dorsum of the foot, which has relatively few sweat glands. This increase seems to be permanent; it has been found to persist for more than a year after operation. It is not however maintained at a constant level but fluctuates through a wide range from day to day. Possibly local changes of pressure, temperature, etc., account for this.

Skin resistance does not depend on somatic nerves, for it does not change when these are cut so long as the sympathetic supply is uninjured. Removal of only the sympathetic nerve-supply to the foot results in an increase of resistance of approximately the same amplitude as when the entire nerve-supply is cut off. Yet this increase is not permanent, as it is under the above-mentioned conditions. According to the author, a possible explanation of this peculiar variation is that muscular activity is still present in the sympathectomised limb.

From observations made on narcoleptic and encephalitic patients it has been found that functional changes in the sympathetic centres of the third ventricle and its vicinity produce an increase in skin resistance, yet it is not comparable to that effected by lesions of the peripheral mechanism. In five patients who exhibited cataplectic phenomena clinically daily palmar skin resistance readings taken when the patients were awake were found to be considerably higher than in normal persons.

J. V.

[72] **Physiological factors involved in the electrical resistance of the skin.**—Curt P. Richter. *Amer. Jour. Physiol.*, 1929, lxxxviii, 596.

An attempt has been made to find an experimental foundation for numerous observations made on the electrical resistance of the body in various normal and pathological conditions, such as sleep, stupor, narcolepsy, etc.

From previous observations it was known that the resistance offered by the body to a galvanic current is localised in the skin. An effort was made

to determine what components of the skin—sweat glands, capillaries, epithelial cells, or cornified cells—control the resistance. It was found that the sweat glands are responsible ; when they are stimulated to hyperactivity the resistance decreases greatly, and it is increased when they are inhibited. This result, however, applies only to the palmar surface of the hand ; the resistance of the dorsal surface is dependent primarily on epithelial cells, and very little, if at all, on sweat glands.

These resistance changes (both palmar and dorsal) can be brought into relationship with the heat-regulating mechanism of the body—the palmar with the sweat glands and sweating, the dorsal with the epithelium and insensible perspiration. They are further correlated with the mental condition of the individual, varying with degrees of tenseness or strain, with sleep, and with normal diurnal changes in mood.

J. V.

[73] **The galvanic skin reflex in normal, thalamic, decerebrate, and spinal cats under anaesthesia.**—G. Wang, J. Pan, and T. Lu. *Chinese Jour. Physiol.*, 1929, iii, 109.

From a series of experiments on cats the authors conclude that there are three nerve centres for the galvanic skin reflex—a cortical, a thalamic, and a spinal centre. The spinal centre consists of cell-groups for sympathetic fibres to the fore- and hind-feet. Their localisation is approximately D4-10 for the former and D12-L3 for the latter.

The thalamic centre is most probably a vegetative centre in the tuber cinereum. The site of the cortical centre remains uncertain.

It seems surprising, according to these experiments, that with higher centres intact the galvanic skin reflex has a shorter latent period and a greater intensity than if these centres are first removed. Perhaps a special mechanism should be assumed in the spinal cord, which makes the lower (spinal) centre inaccessible to incoming afferent impulses under ordinary circumstances and diverts them to higher ones.

The galvanic skin reflex has nothing to do with the vasoconstrictor reflex. The latter is not affected by transections of the neuraxis until the hindbrain is removed, whereas marked changes in the former are observed when the cut is made through the upper border of the midbrain.

J. V.

[74] **The sequel to isolated stimulation and removal of the head of the caudate nucleus in cats** (Folgen isolierter Reizung und Ausschaltung des Streifenhügelkopfes bei der Katze).—R. Ashizawa and F. H. Lewy. *Zeits. f. d. g. exper. Med.*, 1929, lxvi, 157.

Using Clarke's stereotaxic instrument, the authors sought to bring fresh facts to light in connexion with the controversial question of the electric excitability

of the basal ganglia. They repeated older experiments on the head of the caudate nucleus in cats, both before and after degeneration of the pyramidal system.

1. In intact cats they obtained by moderate to strong faradic excitation a combined tonic movement of both sides of the body, limbs (both sides) and tail which they liken definitely to that described by Graham Brown from stimulation of the cross-section of the mesencephalon in decerebrate animals, at a spot corresponding to red nucleus, superior cerebellar peduncle, and posterior longitudinal fasciculus, and quite removed from the pyramid. They consider this movement distinct from any obtainable by pyramidal stimulation; it has the character of a 'Gemeinschaftbewegung.'

2. In cats similar excitation, after complete extirpation of the motor cortex and degeneration of the pyramidal tract, results simply in a minimal motor reaction, so slight as to be negligible, and the authors' conclusion is that "without implication of the pyramidal system excitation of the caudate nucleus does not lead to the above-mentioned series of movements."

They also conclude that "the function of the corpus striatum is to be regarded much less as one of excitation to movement than of influencing and regulation of posture."

At the same time it is not proved that excitation occurs only via the pyramidal system in the case of caudate function, seeing that bilateral removal of the motor cortex in the cat does not greatly affect movement.

S. A. K. W.

[75] **Stimulation experiments on the central pupillary innervation in the corpus Luysii** (Reizversuche zur zentralen Pupilleninnervation am Corpus Luysii).—T. Shinosaki. *Zeits. f. d. g. exper. Med.*, 1929, lxvi, 171.

The author undertook to repeat older experiments of Karplus and Kreidl on the central paths for pupillary innervation, with special reference to the corpus subthalamicum. In eight cats that structure was specifically stimulated with the faradic and galvanic currents and subsequently destroyed (pathological confirmation). The result was as follows: with weak or moderate galvanism (3-5 ma.) the homolateral pupil dilated; with moderate faradism, the pupil contracted, while sometimes the heterolateral pupil slightly dilated. Both currents had also the effect on both eyes (more on the homolateral) of widening the palpebral fissure and protruding the bulbus oculi.

In nine cats the excitation and destruction proved to be in the neighbourhood of the corpus Luysii. No specific reactions were obtained as with direct implication of the corpus, but in six instances bilateral dilatation of the pupil occurred (galvanic current), and in five with the faradic current. In two

cases where the corpus was destroyed directly with a needle, a typical Horner syndome (paralysis of the cervical sympathetic) developed and was demonstrable for at least six weeks, confined to the homolateral side.

In none of these numerous experiments, both excitatory and destructive, was any evidence obtained for a motor function in the corpus Luysii, and the author is convinced that the association of hemiballismus with that body is incorrect ; in his view any disorder of motor function is not caused by lesions of the corpus Luysii but by involvement of motor systems in the vicinity.

S. A. K. W.

[76] **Experimental investigations into the problem of sleep** (Experimentelle Untersuchungen zum Problem des Schlafes).—STURE BERGGREN and ERIK MOBERG. *Acta Psychiat. et Neurol.*, 1929, iv, 1.

THE authors have succeeded in producing somnolence in cats by injecting isotonic solutions of various salts into certain regions of the brain. Their results suggest that the analogous results obtained by Demole are not the effects of calcium chloride itself, as potassium chloride can produce identical results, as can in fact mere acupuncture. The observations of the authors tend to localize more accurately the so-called sleep centres to the nuclei infundibulares and hypothalamici.

M. C.

[77] **The effect of normal and of increased cerebrospinal fluid pressure on the velocity of intracranial blood flow.**—H. G. WOLFF and H. L. BLUMGART. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 795.

IN cats the average ' crude ' intracranial time is 6.05 seconds. This indicates that in normal cats the average true circulation time of the brain is approximately 3 seconds. In normal animals there is a definite relation between the velocity of the intracranial blood flow and the height of the arterial pressure ; the higher the blood pressure is, the faster the blood flow. The velocity of the intracranial blood flow is dependent not only on the height of the blood pressure but primarily on the intracranial arterial-venous pressure difference. If through an increase in intracranial pressure the arterial-venous pressure difference becomes small, the velocity of the intracranial blood flow is slowed. Through cerebral vasodilatation, the flow, though slower, is increased in volume and the circulation remains adequate. However, if the arterial-venous pressure difference becomes so small that the velocity is reduced to approximately half of the normal, the blood pressure rises, the arterial-venous pressure difference increases, and the velocity of intracranial blood flow again approaches the normal. Variation in the systemic arterial pressure and the diameter of the cerebral blood vessels, regulating between them the velocity and volume of the intracranial blood flow, apparently keep constant the internal environment of the brain.

R. M. S

[78] **Transmission of intracranial pressure in hydrocephalus in infancy.**—Kenneth D. Blackfan, Bronson Crothers, and Robert N. Ganz. *Amer. Jour. Dis. Child.*, 1929, xxxvii, 893.

The adult cranium and spinal column are relatively fixed in shape and in capacity, not so the infantile cranium and spinal column. Accordingly variations in pressure readings from the ventricle and the lumbar region are likely to occur in children. The authors by graphic methods recorded simultaneously the pressure in the ventricle and in the spinal theca. They found that the propagation of abruptly applied pressure of short duration from ventricle to spinal space was immediate; and they assumed the probability of widened channels between the two cavities. When delay occurred they suggested narrow channels of communication, but also found this to occur in normal infants. If complete block of propagation of the pressure was present it indicated a complete discontinuity of the channels. From these observations they concluded that such measurements were of possible use in the diagnosis of hydrocephalus and that the application of the 'closed box' theory to infants was unsound.

E. A. C.

[79] **On some systems of fibres in the tegmen of the mesencaphalon** (Su alcuni sistemi di fibre nel tegmento mesencefalico).—U. Poppi. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 59.

The knowledge we have of the systems of fibres in the area of the lemniscus of the pons seems to point to the following facts:

1. In the most caudal section of the pons at the side of the raphe there are descending fibres from the brachia conjunctiva and perhaps also fibres from the predorsal fasciculus.

2. At the same point but further forward appears the temporopontine bundle of the tegmen. This is only visible in the upper part of the pons and rejoins the main bundle at the beginning of the substantia nigra. It originates from the outer third of the peduncle, and runs obliquely to the side of the interpeduncular fossa, whence it becomes fused with the longitudinal fibres of the pons and proceeds to the median nuclei of the pons and perhaps to the reticular nucleus.

3. The frontopontine fibres of the tegmen are erroneously described as a continuation of the frontopallidal bundle. This system is inconstant and its development varies. In the oral section of the pons it is placed more laterally than in the caudal section. It is obvious that in the peduncle it is confused with the main frontopontine bundle.

4. The pallidal systems. In the pons they are only represented by a few fibres which are visible in cases of degeneration of the lemniscus. In the substantia nigra they occupy the lateral dorsal angle in the form of a compact

bundle. The author thinks that this constitutes a direct connection between pallidum and tegmen. He also claims to have established the existence of a subthalamico-peduncular system of fibres.

R. G. G.

[80] **The phylogenetic development of the substantia gelatinosa Rolandi.**—E. KEENAN. *Konin. Akad. v. Wetensch. t. Amsterdam*, 1929, xxxii, No. 4.

CONTINUING his researches on the substantia gelatinosa Rolandi through the animal series, the author summarises the results of his investigations in favour of the view expressed by Ranson, according to which protopathic sensation is associated with the substantia gelatinosa, while epicritic sensation chiefly passes upwards in the cord, by way of the posterior funiculi. In lower animals where protopathic sensation is practically the only kind present the posterior funiculi are small or absent; in mammals, these form no less than 39 per cent. of the total white substance of the cord. Ranson found unmyelinated fibres in the lateral part of the posterior root at the point where the latter enters the cord; they proceed into Lissauer's zone and end in the substantia gelatinosa. Similar unmyelinated fibres have been seen in the descending root of the fifth as well as in its peripheral branches, and they are considered by some to be conductors of protopathic sensibility.

S. A. K. W.

[81] **The diencephalic vegetative mechanisms.**—S. E. JELLIFFE. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 838.

THE author presents a masterly review of the anatomy and physiology of the central vegetative mechanisms. The illustrations have been chosen with care and make it easy for the reader to follow the intricate fibre connections of the diencephalon.

R. M. S.

NEUROPATHOLOGY.

[82] **A contribution to the knowledge of the process of nervous disintegration** (Contributo alla conoscenza del processo di disintegrazione nervosa). —G. B. BELLONI. *Riv. di nerv. e ment.*, 1928, xxxiii, 169.

A REVIEW of the literature is given and the author's experiments are described, bearing on the healing of non-septic wounds of the brain and the influence thereon of certain internal secretions. The neuroglia of the grey matter forms a barrier separating wounded from healthy tissue. That of the white matter shows amœboid transformation and hypertrophy typical of protoplasmic behaviour. The neuroglia does not show proliferation or phagocytosis and

granules may be seen to traverse but not to be formed in its substance. If a lacuna is formed by the wound the glial cells do not fill it up. In animals whose thyroid is removed there is rather more tendency to protoplasmic hypertrophy and amœboid degeneration of the glial cells. Cicatrization and the filling of the cavity form the rôle of connective tissue derived from the pia if the wound is near the surface ; if deeper, a current of fibroblasts may be seen spreading from the margin of the lesion. At the same time a proliferation of vessels is observed which gradually invades the blood clot. This network encloses the necrosed tissue and phagocytosis and absorption take place. The main point of the author's technique is that he is able to distinguish between the neuroglia which proliferates and the microglia which does not. He thinks that reticulate elements of microglial origin have no phagocytic function, but probably represent a regressive form of the hypertrophied microglial elements in which the eventual inclusion of fat particles may lead to fatty infiltration. He concludes that phagocytosis and absorption are the function of mesodermic lymphatic cells of the reticulo-endothelial system.

R. G. G.

[83] **Contribution to the study of heterotopia of the cerebral grey matter** (Contributo casistico allo studio della eterotopia della sostanza cerebrale).—L. AJELLO. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 1.

FULL pathological details are given of one case but the clinical history is not available. The author reviews the literature, and points out that while idiocy and epilepsy commonly accompany the condition it is compatible with perfect mental normality.

R. G. G.

[84] **The question of cerebral air-embolism in cases of attempted abortion** (Zur Frage der zerebralen Luftembolie nach Abtreibungsversuchen).—v. HOESSLIN. *Münch. Med. Woch.*, 1928, lxxv, 764.

AMONG the methods of criminal abortion, the injection of soap and water into the uterus under pressure is one of the more common ; the injection of air at the same time is practically unavoidable. In consequence of this manœuvre serious symptoms and even death not infrequently occur. In some cases the symptoms are chiefly circulatory, in others they are cerebral and take the form of prolonged unconsciousness and epileptiform convulsions.

Air-embolism has been regarded as the cause of the cerebral symptoms in such cases and in the only case fully reported with pathological findings (Neubürger) the histological changes were analogous to those in a case of fat-embolism. The possibility of cerebral *arterial* air-embolism has clinical and experimental confirmation. Symptoms will only arise when the capillary circulation offers a more or less definite obstruction. In one of the recorded post-abortive cases both pulmonary and cerebral symptoms obtained.

When it is a matter of venous air-embolism the explanation is more difficult ; with injection of air into the great veins of the body death results from partial filling of the right side of the heart with air, and the severity of the circulatory symptoms is dependent upon the amount of air injected. If cerebral air-embolism results from entrance of air into the venous system the right heart must be able to overcome the amount of air and the air must pass either through the capillaries of the lungs or through a patent foramen ovale, In experiments on animals it has been found that if air had passed through the pulmonary circulation some was always present in the coronary arteries, and moreover, the lungs and the central nervous system (especially the medulla), suffered damage from the presence of air-containing blood in their capillaries.

It is possible that in cases of abortion the soap may give rise to a corrosive encephalitis.

V. Hoesslin gives a clinical account of a case in which this method of abortion gave rise to severe nervous symptoms—unconsciousness for four days, epileptiform convulsions affecting the left side, and, later, paresis of the right side. The patient made a complete recovery in four weeks. He regards air-embolism—possibly by way of a patent foramen ovale—as the most likely explanation of the symptoms.

J. P. M.

[85] **On the tumours of the ependyma and of the choroid plexus** (Sui tumori dell' ependima e dei plessi coroidei).—F. Battaglia. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 434.

A discussion of the nature of certain tumours. The author concludes that tumours which arise from the choroid plexus retain the appearance of ventricular papillomata. Such tumours have hitherto been confused owing to an imperfect knowledge of the morphological differences normally existing between the two epithelia of the plexus and the ependyma. Nowadays it is possible to distinguish them on cytological criteria. Such criteria recently used by some authors to differentiate fibro-epithelial tumours of a malignant nature arising in the central nervous system seem of doubtful value. It is possible that those tumours which have no connexion with the ventricular cavity have their origin from a relic of ependyma. Other tumours are ascribed to the embryonic tissue of the ependyma and may be justly referred to as spongioblastomata. In a case of the sort here described there may be noticed a double evolutionary orientation, (1) the production of glia cells and (2) of cells having the appearance of epithelium without prolongations. This may represent the attempt of the spongioblasts to develop towards the form of ependymal cells, and may throw light on the neuroepithelial formations met with in certain gliomata. Other epithelial developments which have been described in gliomata may be regarded as true detached ependymal tissue.

R. G. G.

[86] **Dural herniations of the pia-arachnoid.**—Gerald H. J. Pearson. *Acta Psychiat. et Neurol.*, 1929, iv, 97.

At the autopsy of a man dying at the age of 55 from progressive bulbar palsy numerous small dural herniations were noted over the right frontal pole and both parietal eminences. The author puts forward the following suggestion as to their mode of origin: there was first a proliferation and fibrosis of the arachnoid over the whole cortex, which interfered with the absorption of cerebrospinal fluid. The fluid then collected in the pial meshes and by pressure-irritation caused further fibrosis and thickening of the membranes. Weak points in the upper layer of the pia and arachnoid gave way and the underlying layers herniated through them to form papillomatous projections. Where these projections were subjacent to a weak spot in the dura or where their size produced a dural atrophy, the herniation extented through this membrane.

M. C.

[87] **The pathogenesis of osteoarthropathy in paralysed limbs consecutive to trauma of the spinal cord** (La patogenesi delle 'osteo-artropatie e delle para-osteo-artropatie negli arti paralizzati in seguito a traumi del midollo spinale).—E. Repetto. *Il Cervello*, 1929, viii, 53.

A number of different theories have been advanced to account for the development of arthropathic change in limbs paralysed after spinal trauma.

The author has conducted a series of experiments in rabbits, sectioning the spinal cord and endeavouring to produce arthritic lesions by various procedures. He examines in turn the theories of abnormal trophicity, pathological innervation, infection, toxicity, arteritis of joint vessels, pathological calcification, and vasomotor or spmpathetic disorder, and dismisses them all as a result of his experiments. His conclusion is that intra-articular trauma of periosteum leads to post-paralytic osteo-arthropathy, and trauma alone. In his experimental animals it was produced by the galvano-cautery.

S. A. K. W.

[88] 1. **Nitrogenous metabolism in postencephalitic rigidity.**
2. **Creatine and rigidity.**—Martin Hirst and A. G. Imrie. *Quart. Jour. of Med.*, 1927, xxi, 139; 1928, xxi, 401.

In their first paper the authors found that a mild degree of rigidity did not influence the output of uric acid but that in a severe case the output was high. If the rigidity was reduced by hyoscine the uric acid was correspondingly diminished. In all cases creatine was excreted. In their later communication creatinuria was found to occur constantly in the marked degrees of Parkinsonian rigidity. They suggest that there is an association between the activity of the muscles in this pathological state and the amount of uric acid and creatine excreted.

E. A. C.

[89] **Glycolysis in cerebrospinal fluid and its clinical significance.**—KATHLEEN CHEVASSUT. *Quart. Jour. of Med.*, 1927, xxi, 91.

THE necessity for an early quantitative examination of the sugar content in the cerebrospinal fluid is stressed; and observations are recorded showing the influence of the length of time between withdrawal and estimation of sugar. A progressive decrease in the sugar content is demonstrated, which diminution is presumed to be due to a glycolytic ferment present in the fluid. It would have been of interest to have had the cellular content of the fluids, as it is possible that the ferment may be the result of the presence of the cells and that it is not an inherent part of the fluid itself. Attention is also drawn to the fact that ventricular fluid contains more sugar than either the cisternal or lumbar fluid. The paper is one of interest and raises many problems regarding the chemistry of the cerebrospinal fluid.

E. A. C.

[90] **The colloidal reaction of Takata and Ara** (La reazione colloidale di Takata e Ara).—G. UGUCCIONI. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 306.

THIS reaction is carried out as follows. Three reagents are required: carbonate of soda solution 10 per cent., sublimate solution ·5 per cent., and fuchsin (Grubler, non acid) ·02 per cent. To one c.c. of spinal fluid is first added a drop of the sodium solution, then with a graduated pipette ·3 c.c. of a mixture in equal parts of the sublimate and fuchsin solutions. The reaction is simple and purely chemical, and permits a differentiation with a single dilution between inflammatory conditions of the central nervous system with increase of the total albumen, and degenerative processes with increase of the globulin only.

It cannot be used as a substitute for the Wassermann reaction but it is sensitive to though not specific for neurosyphilitic lesions, especially parasyphilis. In general paralysis treated by malaria and arsenical preparations this reaction, like the Wassermann reaction, may become negative.

R. G. G.

[91] **Some observations on the leucocyte count in epilepsy.**—H. A. PATTERSON and S. M. WEINGROW. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 412.

THE following conclusions were based on a study of 182 epileptics. The total white cell count in epilepsy is widely variable: 52 per cent. of the cases showed leucocytosis; 25.5 per cent. gave normal counts, and 20.3 per cent. showed leukopenia. The inconstancy of the blood picture in epilepsy, together with the relatively few cases on which frequently the conclusions previously reported are based, perhaps accounts for the conflict of opinions in the literature. The leucocytosis observed by the authors was not neutrophilic but lymphocytic.

Definite evidence of epileptic leucocytosis being influenced by digestion of food was lacking. Within the limits of physiological variation, the rise in the leucocyte count is the same in non-idiopathic as in idiopathic epilepsy.

A remote effect is not produced by either grand or petit mal seizures on the white cell count in both idiopathic and non-idiopathic epilepsy. It seems that the leucocytosis frequently encountered in epilepsy is not due to the disease per se, but to some concomitant phenomenon. A connection between the leucocyte count in epilepsy and the presence or absence of an aura apparently does not exist. Observations failed to show any relation between onset and duration and the white cell count in epilepsy. The leucocyte count in epilepsy does not appear to be affected by the secondary anæmia which often accompanies this condition.

R. M. S.

SENSORIMOTOR NEUROLOGY.

[92] **Vasomotor trophic changes in cerebral arteriosclerosis** (Vasomotorisch-trophische Veränderungen bei zerebraler Arteriosklerose).—W. SPIELMEYER. *Monats. f. Psychiat. u. Neurol.*, 1928, lxviii, 605.

THIS paper is an important contribution to our knowledge of the pathogenesis of transient cerebral attacks and also of cerebral softening in that it illustrates the trend of modern thought as applied to an important problem in cerebro-vascular disease. The text of Spielmeyer's article is that pathologically demonstrable areas of ischæmia can exist in the brain unassociated with adequate anatomical changes in the blood-vessels of that part. It is suggested that there is a functional disturbance of the blood-supply due to a vaso-constriction of the arteries and capillaries lasting a longer or shorter time. The clinical expression takes the form of either transient or permanent focal manifestations. Spielmeyer points out that the ischæmic zones may appear in stained preparations as a mere bleaching of the cortex: these may be overlooked on cursory examination and herein possibly lies the explanation of those cases of hemiplegia which are recorded as having no demonstrable pathological cause. It is interesting to note how the clinician—who has been long misled by the negative findings of the physiologist—is gradually returning to the conception of a vasoconstrictor process in the cerebral blood-vessels as a pathogenic agency.

M. C.

[93] **Vascular spasms in neurology** (Les spasmes vasculaires en neurologie).—F. BREMER. *Revue d'Oto-Neuro-Ophtalmol.*, 1928, vi, 297.

A USEFUL review of the whole question of angiospasm in regard to the nervous system leads to the following generalisations.

1. The etiology of angiospasm is diverse; it includes local arteritis (not altogether definite), toxic agencies, endogenous or exogenous; and constitutional spasmophilia (Raynaud's disease, migraine).

2. The pathogenic role played by the vasomotor system is doubtful, largely because of the insignificance of vasomotor innervation of cerebral vessels, especially in their terminal branches. Exogenous and endogenous poisons alike probably act directly on smooth muscle fibres. Some paroxysmal angiospasms are perhaps the expression of anaphylaxis.

J. S. P.

[94] **The clinico-anatomical syndrome of lesions of the anterior choroid artery** (La sindrome anatomo-clinica conseguente a lesione dell'arteria coroidea anteriore).—U. Poppi. *Rivista di Neurol.*, 1928, i, 466.

Clinically, a lesion of the anterior choroid artery reveals itself by (1) hemiplegia, (2) hemianæsthesia, (3) lateral homonymous hemianopia, (4) vasomotor change (including œdema), (5) the 'thalamic' hand. The artery supplies the posterior limb of the internal capsule, the thalamus (in part), the globus pallidus (in part), and also the external geniculate body and part of the anterior commissure.

S. A. K. W.

[95] **Congenital aneurysm of the cerebral arteries.**—F. H. K. Green. *Quart. Jour. of Med.*, 1928, xxi, 489.

A careful histological examination of the wall of each of five aneurysms found in three cases, has been carried out; this has included the examination of serial sections. In one, the smallest aneurysm, a definite localized defect was found in the muscular and elastic coats. As no syphilitic, mycotic, or traumatic origin could be found, the hypothesis of Eppinger that these aneurysms are congenital was apparently upheld and confirmed by histological examination. Additional confirmation of this was present in one case, when other vascular maldevelopments were found, such as co-arctation of the aortic isthmus, a bicuspid aortic orifice, and fenestration of the mitral cusps.

E. A. C.

[96] **Decerebrate rigidity and tonic reflexes of the neck during apoplectic coma. Mesencephalic lesions recognized during life** (Ipertonia da scerebrazione e reflessi tonici dal collo durante il coma apoplettico. Lesione mesencefalica riconosciuta in vita).—V. M. Buscaino. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 106.

A case of the above is described, with post-mortem confirmation. The author seemingly thinks it the first case in the literature showing Sherrington's phenomena of decerebration and the tonic neck and limb reflexes of Magnus and da Kleijn observed during apoplectic coma. It demonstrates the possibility of diagnosing from the above symptoms the participation of the upper

part of the brainstem in the lesion, as well as of the occurrence of the reflexes of Magnus and de Kleijn without the retention of the wholly or partially intact sensorium. A certain degree of rigidity is necessary before these reflexes can be demonstrated.

Pyramidal lesions are not, as some think, essential for the production of these symptoms, but lesions of the basal ganglia alone without involvement of the pyramidal tracts cannot cause them. Ordinarily such lesions do not produce tonic reflexes in apoplectic coma and this only occurs if the brain stem is also involved. Experimental work places the lesion in the region of the red nucleus. The case shows that when the tendon reflexes are maintained in deep coma the arc does not go through the cortex ; their preservation probably depends on the same medullary centres as are responsible for decerebrate rigidity and these have much the same localisation in man as in animals. Finally, early contracture at the time of a stroke probably depends on the same lesions in the brainstem.

R. G. G.

[97] (1) **Extrapyramidal movement disorders in advanced age** (Extrapyramidale Bewegungsstorungen im höheren Alters).—S. S. Freund and R. Rotter. *Archiv f. Psych.*, 1927, lxxxi, 751.

(2) **On extrapyramidal diseases of old age with a contribution to the pathogenesis of senile changes in the parenchyma** (Ueber extrapyramidale Erkrankungen des höheren Alters mit einem Beitrag zur Pathogenese seniler Parenchymveränderungen).—C. S. Freund and R. Rotter. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, cxv, 198.

In these two valuable contributions the authors have reported in anatomical and clinical detail eight examples of an akinetic syndrome associated with senility. The cases are considered in three groupings : the first subdivision includes cases illustrating a hypertonic-hypokinetic symptom-complex of the arteriosclerotic-rigidity type. The putamen was the seat of severest pathological change although lesions were demonstrable throughout the corpus striatum and also in the capsule of the olives and in the nucleus dentatus. To the second group are relegated those cases which were characterized clinically by a combination of rigidity and hypotonia. Pathologically the cases proved to be of the nature of a combined strio-pallido-dentato-rubro-peduncular system disease, in which the nucleus ruber was ' isolated ' (in the sense of Munk) from both afferent and efferent influences. Group 3 contained two cases in which cortical or subcortical lesions co-existed and were notable clinically on account of the added manifestations of apraxia, agnosia, echolalia, and by marked psychical changes. The second of the two patients belonged clinically to the cadre of Jakob's ' senile Vorsteifung.'

In their second paper the authors review in interesting fashion the pathophysiology of the motility disorders of advanced age. A valuable discussion

centres round their sixth case, in which a state of chronic deforming arthritis was associated with a Parkinsonian syndrome. The correlation of these two morbid states is fully discussed and the authors conclude that joint deformities of cerebral (extrapyramidal) origin do occur; in outward appearance they exactly resemble the changes of peripheral causation met with in chronic progressive polyarthritis.

M. C.

[98] **The blinking reflex in hemiplegia.**—HOLGER EHLERS. *Acta Psychiat. et Neurol.*, 1929, iv, 47.

THE author has studied in a series of cases of hemiplegia the blinking reflex excited by the trigeminal (contact with the cornea or conjunctiva) and the blinking reflex of optical stimulation (strong light stimulus or threatening visual impressions).

Cases with hemianopia or corneal insensitivity were excluded from this study. The results in the other patients were complex; in some cases both types of blinking reflex were diminished in the homolateral eye. In other instances, only one of the reflexes was reduced, while in a few cases the blinking reflex for light was exaggerated. It is tentatively suggested that the optical blinking reflex may have a duplex character with distinct anatomical bases; thus the blinking reflex for light stimuli is probably elicited via the brainstem only, while the blinking response to threatening danger entails the implication of the visual cortex.

A practical point arises from this study; the usual practice of testing for hemianopia in a semiconscious or stuporose patient by means of threatening gestures towards the eyes from various angles is thus shown to be an untrustworthy measure. Loss of the usual blinking response does not necessarily mean hemianopia, and, conversely, lively optical blinking responses have been noted in cases with hemianopia.

M. C.

[99] **Unusual hyperkinetic symptoms in connexion with the voice: megaphonia, hyperodia** (Ungewohnliche hyperkinetische Symptome auf dem Gebiete der Stimme: Megaphonie, Hyperodie).—J. WILDER. *Monats. f. Psychiat. u. Neurol.*, 1929, lxxi, 373.

UNUSUAL complications of epidemic encephalitis in respect of the voice and of speech functions generally have frequently been noted, but those here reported are rare if not unique. In the first case the patient exhibited an irresistible impulse to sing, and, in less degree, to read aloud; in the second case, the impulse was to speak in a much louder voice than usual (this was not, however, an encephalitic but an arteriosclerotic case).

The suggestion is made that the hyperodia signifies the release by disease of a "primitive psychomotor automatism," though what is meant by this term is not explained; the megaphonia is attributed to a release of function of both a pyramidal and an extrapyramidal component in voice production.

S. A. K. W.

[100] **The distribution of motor apraxia in the segments of the body** (Zur Frage der Verteilung der motorischen Apraxie auf die Körperteile). —O. Maas and O. Sittig. *Monats. f. Psychiat. u. Neurol.*, 1929, lxxiii, 40.

It was long ago observed by Hughlings Jackson that certain motor phenomena were prone to develop in one part of the body rather than another. The usual sites for the commencement of Jacksonian epilepsy are forefinger and thumb, face, and foot (great toe). Similarly, in hemiplegia those muscular groups are most severely affected whose function is 'most voluntary,' and those least affected whose function is 'most automatic.' Again, in cases of 'phantom limb' after amputation those parts mostly take a share in the phantom residuum which have most small muscles (hand, foot), while more centrally situated groups participate little or not at all.

The authors apply these generalisations to the question of motor apraxia and conclude from their observations that the same rules apply in respect of that syndrome; isolated apraxia develops in the upper extremities, or in the face; if it implicates the whole body musculature then it is more pronounced in specific groups, as above, than in others. Clinical cases are cited in support of their conclusions.

S. A. K. W.

[101] **An unusual form of meningitis in childhood, excited by the Koch-Weeks bacillus** (Eine eigentümliche Meningitisform im Kindesalter, hervorgerufen durch Koch-Weekssche Bazillen).—Meyer and Steinert. *Münch. Med. Woch.*, 1928, lxxv, 945.

The authors refer to a paper by Hugo Meyer in which attention was called to the prevalence of a peculiar form of meningitis in children, the exciting cause of which was the Koch-Weeks bacillus. They themselves record three cases, with two autopsies.

The patient in the first case was a little girl of twenty months, who had been febrile for four or five days before symptoms of meningitis were apparent; then followed rigidity of the neck and Kernig's sign, somnolence and unconsciousness, and finally tonic and clonic spasms. Death occurred on the seventeenth day of the illness. Cerebrospinal fluid findings:—fluid under increased pressure; turbid; dense fibrin clot after long standing; cells, 1800 per c. mm. (polymorphs); Nonne and Pandy reactions strongly positive.

The second case was that of a male infant, seven months old, whose illness began with high fever and convulsions ; three days later, he was admitted to hospital with the clinical appearances of ' Broncho-tetanie ' ; the first meningitic symptoms were seen on the sixth day of the illness and death occurred three days later.

The fluid findings were exactly similar to those in the previous case.

The third patient, a little girl of two years, began her illness with vomiting and a rise of temperature. A few days later neck rigidity and Kernig's sign were present and the spinal fluid was turbid. A month after the beginning of the illness convulsions began and consciousness was lost ; death did not occur till the end of the seventh week. The temperature was never high, ranging between 38° and 39° C.

In all three cases bacteriological examination of the cerebrospinal fluid by smear and culture revealed organisms having the characters of the Koch-Weeks bacillus.

Autopsies were held in the first two cases ; pus was abundant and poured out when the skull was opened. Patches of pressure atrophy were present in the dura. The leptomeninges were inflamed, with occasional circular hæmorrhages ; over the anterior and lateral parts of the base of the brain and over the posterior parts of the convexity the leptomeninges were covered by a thick layer of viscous clinging pus of a striking yellow-green colour ; it lay over the surfaces of the gyri as well as in the sulci, thus reproducing the brain-contours ; the gyri were flattened. The ventricles were dilated and filled with turbid fluid. In the second case the mucous membrane of the nose and ethmoid sinus was inflamed and swollen, had hæmorrhages within it, and was covered with purulent yellow mucus. In Case 1 the left middle ear also contained yellowish pus. Smears taken at the autopsies revealed the same organism as had been found in the spinal fluid.

It may be that the preliminary clinical stage before the appearance of symptoms of meningitis corresponds to the development of the infection in the nasal passages. J. P. M.

[102] **Amaurotic family idiocy and general lipoid degeneration.**—B. SACHS. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 247.

SACHS reviews the pathology of amaurotic family idiocy in the light of recent research. His paper contains many points of interest which deserve careful study.

A few years ago Bielschowsky insisted that the pathogenesis of this disease must be linked with the source of lipoid substance within the nerve cells. Acceptance of this view implied opposition to the conclusions of Schaffer, who supposed that the lecithinoid granules found with startling uniformity in all the nerve cells of the central nervous system were to be regarded as products of degeneration. In the meantime, clinical and pathological studies

have revealed a surprising relationship with another group of diseases—with Gaucher's disease and with a familial and fatal disease of infancy showing great predilection for the Hebrew race and bearing some, if not complete, resemblance to Gaucher's disease. This infantile form is a lipoid cellular splenohepatomegaly or lipoid histiocytosis, and is now known as the Niemann-Pick type, in contradistinction to the Gaucher type. Both types would appear to be variants of a general lipoid cellular degeneration with enlargement of the spleen and liver. The Gaucher type may occur at any age. The Niemann-Pick type terminates fatally before the end of the second year. In the Gaucher type, the lipoid invasion affects the reticular cells and the connective tissue cells, the so-called histiocytes. In the Niemann-Pick type, the same invasion occurs not only in the entire reticulo-endothelial apparatus, but also in the elements of all other body tissues—in the muscles, in the glia, in the ganglion cells and in all epithelial cells, even in the cells of cartilage. Sachs suggests that if the similarity of these two types is to be acknowledged, the Gaucher type might be called the juvenile, and the Niemann-Pick type the infantile form of this splenohepatomegaly, thus emphasising the resemblance of these forms to the two types of amaurotic family idocy. The underlying disease process in all these forms is evidently a profound and primary disturbance of metabolism and it is Pick's contention that in Tay-Sachs disease the same disturbance in lipoid metabolism is the predominent factor. When this metabolic disturbance affects the entire body, the Niemann-Pick type, a universal disturbance of lipoid metabolism, is developed; the Tay-Sachs type is a disturbance of the same kind restricted to the nervous system.

In his last publications Pick recorded several cases of the Niemann-Pick type in which he made a complete examination of all the organs, including the central nervous system: he also recorded that in one of these cases, easily recognised as a form of the Niemann-Pick type, all of the cardinal symptoms of the Tay-Sachs type were present. Before deciding whether or not one is to accept the relationships of amaurotic family idocy to the Niemann-Pick type, it will have to be shown that the symptoms of amaurotic family idiocy occur in a relatively large number of cases of the Niemann-Pick type, and it will be still more important to examine all the organs of a child dying of amaurotic family idiocy to prove that in those cases the histological cellular changes are similar, as Pick believes, to the cellular changes that are now so well known as occurring in every part of the central nervous system.

R. M. S.

[103] **Latent syphilis in post-traumatic complaints** (Latente Lues bei post-traumatischen Beschwerden).—D. Weisz. *Deut. Zeits. f. Nervenheilk.*, 1929, cvii, 260.

The time-honoured controversy as to the influence of trauma on syphilis of the nervous system is here again discussed and illustrated by a number of cases.

In the author's experience exacerbations of existing neurosyphilitic symptoms following trauma—even if the latter has been relatively severe—do not persist. The chief result of such traumata is the development of general nervous symptoms. In four cases in which local symptoms arose, two responded to treatment; in the third case the trauma followed neurosyphilis, and in the fourth the symptoms of the two processes were independent of each other.

Latent syphilis can light up spontaneously at any time, as well as in consequence of trauma. The usual effect of trauma is to produce a temporary exacerbation of meningeal processes. Cases with obvious existing syphilitic symptoms and with only latent syphilis react in the same way where trauma is concerned; hence it is well not to overestimate the effect of the latter in the production of syphilitic symptoms.

J. S. P.

[104] **Amyotrophic lateral sclerosis or bulbospinal myasthenia with exaggerated tendon jerks and fibrillation** (Sclérose latérale amyotrophique ou myasthénie bulbo-spinale avec exaltation des reflexes tendineux et contractions fibrillaires).—L. Van Bogaert and J. Van den Broeck. *Jour. de Neurol. et de Psychiat.*, 1929, xxix, 380.

A clinical presentation of an unusual set of symptoms occurring in a young man. The outstanding features were:

1. Bitemporal pallor of the optic discs with conservation of vision.

2. Pupillary inequality; ptosis; paralysis of the pterygoids and masseters; bilateral facial palsy; nerve deafness; paralysis and loss of sensation of the palate and pharynx. Atrophy and fibrillation of the tongue.

3. Generalized wasting, especially the peroneal and scapular groups, with fibrillation. Reaction of degeneration.

4. Exaggeration of the tendon jerks with right-sided ankle clonus and Babinski response.

There was no abnormality in the spinal fluid and the Wassermann reaction was negative.

M. C.

[105] **A contribution to the pathology and etiology of Landry's paralysis** (Beiträge zur Pathologie und Aetiologie der Landryschen Paralyse).—Holländer and Karoliny. *Münch. Med. Woch.*, 1928, lxxv, 1549.

In 1859 Landry described a clinical picture, characterised by flaccid paralysis of all limbs coming on quickly and with distinctive sequence, the paralysis affecting first the lower extremities, then the upper, then the muscles innervated by the cranial nerves and finally the diaphragm; disturbances of sensation are absent or at any rate slight. In this paper two cases approximating to the Landry type are recorded with post-mortem findings. The first was that of a man of 26; the symptoms began with numbness in the toes of the right

foot and paralysis gradually spread upwards, the legs being completely paralysed by the sixth day; by the ninth day the facial muscles were weak and by the eleventh swallowing had become difficult; arm movements were at that time still possible. All tendon jerks were absent. There was no disturbance in micturition and defæcation. Sensation was somewhat impaired generally over the lower limbs, and on the outer sides of the legs there was definite loss of appreciation of touch, painful stimuli and temperature. On the fifteenth day of illness paralysis of the diaphragm was apparent, and on the following day the patient died of bronchopneumonia. Post-mortem the findings were those of an acute infection; the spleen showed acute enlargement; histologically there were numerous inflammatory foci round the vasa nervorum, and the interstitial tissues of the nerves were swollen and œdematous; the myelin sheaths showed granular degenerative changes. The changes within the nerves were particularly noticeable in the proximal parts, but in the spinal cord no distinctive changes were found. In the nerve sections numerous Gram-positive encapsulated diplococci were discovered in the foci; they were apparently pneumococci and the authors of this paper regard them as the cause of the polyneuritis.

In the second case, the patient, a young doctor of 25, died on the thirteenth day. His symptoms had begun with pains in the legs and back and with fever. The Wassermann reaction was strongly positive. On the fifth day the legs began to feel numb and by the ninth day the legs were completely paralysed; the arms were weak and the face was paralysed on the right side. The next day all limbs were paralysed and passive movement of them caused pain; the reflexes were all absent. No satisfactory examination of sensation could be made. On the twelfth day the pharynx and diaphragm became paralysed. Post-mortem there were found septic bronchitis and tonsillitis. Histological examination demonstrated perivascular inflammatory foci in the nerves and in the brain and other organs, the process being one of extensive distribution. As in the previous case, Gram-positive diplococci were found in great numbers.

While the clinical course of these cases in many ways resembled that of Landry's paralysis, we think they would be better described in view of the histological findings as acute polyneuritis. Though the limits of the category of 'Landry's paralysis' are still not definitely laid down, the tendency is to restrict the term to cases without sensory involvement and showing after death changes in the anterior horn cells, but little or no inflammatory reaction. At the same time, the cases here recorded are of interest and the presence of organisms within the inflammatory foci is important. J. P. M.

[106] **Extramedullary tumour; arachnitis fibrosa cystica et ossificans; gliosis of the medulla.**—FRANCIS HARBITZ and INGVAR LOSSIUS. *Acta Psychiat. et Neurol.*, 1929, iv, 51.

A FEMALE, age 26, had a period of diplopia and weakness of the arms lasting several months, ten years before coming under observation. Two years before

admission her legs became stiff and weak. On examination a spastic paraparesis was found with nystagmus and intention-tremor of the extremities ; the tongue deviated on protrusion. The changes in the spinal fluid indicated compression. Later she complained of headaches ; papillœdema developed, and cutaneous sensibility became lost below D 6. At autopsy the following changes were noted :

1. A large endothelioma behind the medulla and upper cervical cord.
2. Chronic arachnitis fibrosa cystica in the region of the seventh and eighth thoracic segments.
3. A gliomatosis with central necrosis in the sixth and seventh cervical segments.
4. Numerous osseous lamellæ imbedded in the arachnoid.

M. C.

[107] **Tumours of the spinal cord in childhood.**—Byron Stookey. *Amer. Journ. Dis. Child.*, 1928, xxxvi, 1184.

This comparatively uncommon disease has aroused interest recently and the author records eight such cases. He reviews the symptomatology and points out the late onset of sphincter disturbance ; he lays stress on the value of manometric readings at lumbar puncture for the early diagnosis.

E. A. C.

[108] **Intramedullary cyst of the spinal cord associated with a circumscribed intramedullary tumour.**—C. E. Dowman and W. A. Smith. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 583.

Intramedullary cysts may occur either above or below a tumour of the spinal cord as a result of cystic degeneration of the tumour, softening of an associated central gliosis, dilatation of the central canal, or foci of necrosis from vascular changes at a distance from the tumour.

In the author's case the cyst involved the entire cervical cord and was found above and separate from an intramedullary sarcoma. It was under marked tension and large enough to produce a spinal block. The tumour was diagnosed as a sarcoma but may possibly have been a hæmangioma or a hæmangioblastoma.

With regard to the mechanism of the cyst formation, it was not found possible to draw any definite conclusions. There was no indication that it was caused by cystic degeneration of a neoplasm, and a cyst of this size and under such tension could hardly be of the type associated with a gliosis. Whether due to a dilated central canal or to necrosis from distant circulatory changes, it appeared probable that the cord tumour caused a stasis of cerebrospinal fluid and contributed to the formation of the cyst and the tension of the fluid within it.

R. M. S.

[109] **Contribution to the clinical and pathological study of the syringomyelia syndromes** (Contributo allo studio clinico e patogenetico delle sindromi siringomieliche).—G. VERCELLI. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 21.

THREE cases are fully described and the literature is reviewed. The author concludes that in syringomyelia the importance of trauma is subordinate to the predisposition in the tissues, which may be either congenital or acquired. Vertebral deviation is important since this may be for a long time the only symptom of a developing syringomyelia. It may be due to an osteoarthritic atrophy or to muscular weakness causing distortion.

The total anæsthesia frequent in syringomyelia may be due either to a lesion of the posterior columns of the cord or to one in the grey matter which may occur at a considerably higher level. There may be multiple lesions irregularly distributed: the frequency of their unilateral character points to a congenital origin.

Deep radiography is the only therapeutic measure available to-day and acts by its power of fixing and resolving gliomatous processes.

R. G. G.

[110] **Myelomalacia of rapid development, following antityphoid vaccination** (Myélomalacie à évolution foudroyante, survenue après une vaccination antityphique).—DIVRY, MOREAU and ORY. *Jour. de Neurol. et de Psychiat.*, 1929, xxix, 369.

FOUR to five days after an inoculation with antityphoid serum, a man of 21 developed a quadriplegia which became complete in the course of 24 hours. The patient died of respiratory failure 60 hours later. Autopsy revealed complete necrosis of the spinal cord mainly in the region of the cervical enlargement. There were marked vascular anomalies in the nature of hæmorrhages and thrombosis, but no signs of neuroglial reaction. The authors review the literature dealing with the neurological complications of antityphoid inoculation.

M. C.

[111] **Hemitetany, with a discussion of the site of the spasms** (Zur Klinik der Hemitetanie mit Erörterungen über den Auslösungsort der tetanischen Krampfe).—E. REDLICH. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1929, cxx, 236.

THE two cases herein described are of considerable interest, seeing that they belong in reality to the cervical variety of disseminated sclerosis, and present the usual symptom-complex of that particular group. In addition, however, typical tetany-like seizures occurred (in the first case) mainly in the right arm and hand, but at first also in the left. Trousseau's phenomenon was positive. An elastic bandage round the right arm brought out tetanoid spasms in a few

seconds, as did pressure on the ulnar nerve. Yet other usual manifestations of tetany were awanting. The calcium content of the blood was found on two occasions to be within normal limits. More or less identical clinical observations were made in the second case.

The author examines in turn various theories to account for the peculiarities of his two cases. He has not found a similar combination of unilateral tetany with cervical cord symptoms in other recorded instances of so-called cervical disseminated sclerosis. He notes in particular the absence of any assignable cause for the tetany and of any of the usual accompaniments. After a consideration of several analogous cases of tetany accompanied by symptoms of involvement of the central nervous system, he concludes (as the reviewer thinks, on inadequate grounds) that "the site of origin of the hemitetany in our two cases lies in the subcortical ganglia"; and further that "the disorders both of deep sensibility and of tactile gnosia are referable to lesions in the subcortical ganglia and the internal capsule."

S. A. K. W.

[112] **The sciatic syndrome.**—M. GROSSMAN and M. KESCHNER. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 398.

THE term sciatica, as it is generally employed, is misleading. It tends to direct attention away from the most common and remediable underlying cause of the various clinical conditions characterised by pains in the lower part of the back and limbs. The use of this term should therefore be discontinued, and an effort be made to determine the underlying cause and to designate the condition accordingly, and as being associated with the sciatic syndrome.

In the absence of a polyneuritis or polyradiculitis from whatever cause (toxic, infective, metabolic, constitutional, vascular, etc.), a primary mononeuritis or radiculitis of the sciatic nerve is extremely rare. In practically all the authors' cases the sciatic syndrome was secondary to some pathological process in osseo-arthritic structures or their contiguous soft parts in the region under discussion. Cases in which the patients have paroxysmal attacks of lancinating or shooting pains in the lower part of the back and in the lumbosacral distribution without objective evidences of involvement of the lumbosacral roots or nerves, or of the osseo-arthritic structures or their adjacent soft parts (muscles, tendons, fascia, etc.) in this region should be designated as "sciatic neuralgia." The presence of only three such cases in a series of 317 indicates the rarity of this condition, notwithstanding that most authors emphasize its frequency and employ the term 'sciatica' as if it were synonymous with 'sciatic neuralgia.'

By far the largest number of patients presenting the sciatic syndrome show demonstrable evidence of involvement of the osseo-arthritic system or of the

contiguous soft parts in this region. This group may be divided into two subgroups: (*a*) Cases without demonstrable signs of organic nervous disease. In the absence of such signs, these cases present an orthopædic problem, and for convenience they may be designated 'orthopædic cases.' (*b*) Cases with demonstrable evidence of involvement of the lumbosacral roots or nerves. Depending on the nature and distribution of the involvement of the nerve they may be designated as cases of lumbar, lumbosacral or sacral radiculitis. This group included 64 per cent. of the authors' material.

Except in such rare cases as sacralisation of the fifth lumbar vertebra, calcification or ossification of the soft parts, or in primary or metastatic neoplastic or tubercular processes in the spine or pelvis, positive X-ray observations of spondylitis or arthritis are not conclusive as to the etiological relationship of these observations to the sciatic nerve.

R. M. S.

[113] **Motor phenomena in sciatica.**—D. A. Schambouroff. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 392.

In papers devoted to sciatica attention is usually concentrated on the sensory phenomena, and it is often assumed that neuralgia of the sciatic nerve leaves the motor functions entirely or almost entirely undisturbed. In reality, this is not true, for in this disease there is a whole series of motor phenomena which are of great diagnostic importance. Of these the Kernig sign is the best known. The Lasègue contralateral sign, often confused with the first-named, consists of the following: the raising of the unaffected stretched leg brings on pain on the affected side, mainly in the ischiatic area, sometimes in the lumbar region, and sometimes down the entire leg. Flexing the head (the Lindner sign) occurs as follows: if enforced passive flexion of the head to the chest is effected in a recumbent or sitting posture with outstretched legs, pain arises in the lumbar region, in the ischiatic area, and sometimes down the entire leg on the affected side. In acute cases this symptom is clearly manifested in a recumbent posture; in subacute cases it may fail to appear, but it will do so if the patient is made to sit up in bed with the legs outstretched. Closely allied to the Lasègue sign is Neri's sign of bowing. When the patient, in a standing posture, is made to bow, he flexes the knee on the affected side as if curtseying; simultaneously a sensation of pain appears in the leg. The author concludes that these phenomena are of a reflex nature and are immediately connected with the pains arising from tension and irritation of the nerve and roots. Besides this, the immediate irritation of the motor parts of the roots apparently plays a part in their origin.

R. M. S.

[114] **Unilateral neurofibromatosis of the cranial and deep cervical nerves.**—G. T. Pack. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 919.

In the case reported there was an enlargement of the left side of the face due

to a myxomatous condition of the soft parts ; the bony framework was actually smaller on the affected side than on the normal. In the left upper eyelid, the left cheek and the left side of the neck were numerous discrete, circumscribed, freely movable, painless nodules. These tumours were composed of nerve fibres and connective-tissue cells. The local interstitial proliferation of fibroblasts constituted the major portion of the tumour and dissociated the nerve fibres.

R. M. S.

[115] **Juvenile acrodynia.**—C. W. Wychoff. *Amer. Jour. Dis. Child.*, 1929, xxxvii, 88.

Twelve further cases of this disease are added to the literature. All the cases completely recovered so no histological investigation was possible. The suggestion made by Greenfield and Paterson that the disease is a polyneuritis has been upheld, as in all cases there was a diminution in muscular power and sensation peripherally : no case, however, showed the inability to hold the head erect as described by the British authors. No common etiological factor was found ; but the author considers that the condition may be infective (following infections of the respiratory apparatus) and not of the nature of deficiency disease.

E. A. C.

[116] **The neurological aspects of intermittent claudication.**—T. K. Davis. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 571.

Though intermittent claudication may be a symptom of any form of arterial disease, usually it is due either to arteritis obliterans or to arteriosclerosis of all or a portion of a peripheral vessel. Davis reports ten cases in which the condition was a symptom of arteriosclerosis. Only two of the ten were women—a fact which emphasises the well-known greater occurrence of the condition in men. Transitory neurological signs in the precise form described by Dejerine were not found, but it was possible to distinguish two outstanding and contrasting spinal syndromes : (1) a predominantly spastic paraplegic syndrome, and (2) a predominantly poliomyelitic syndrome. In each there was a moderate involvement of the posterior columns, as evidenced by a disturbance of vibratory sensation. Impairment of bladder control was present and was conspicuously absent in another with a poliomyelitic syndrome of severe type.

R.M.S.

[117] **The clinical syndrome and pathological histology of periarteritis nodosa** (Zur Klinik und pathologischen Histologie der Periarteritis nodosa).—J. Silbermann. *Monats. f. Psychiat. u. Neurol.*, 1929, lxxii, 225.

The rare disease known as periarteritis nodosa is commonly considered to be clinically recognisable by the following triad of symptoms : (1) a chlorotic

marasmus; (2) polyneuritis and polymyositis of varying intensity; (3) symptoms referable to the intestinal tract (abdominal pains, diarrhœa, vomiting, melæna, perforation and peritonitis). In addition, frequent slowing of pulse, albuminuria, and fever deserve mention.

An interesting case of this kind is described in full: the condition of obscure polyneuritis was eventually diagnosed during life as being due to periarteritis and the surmise corroborated by pathological examination. In the central nervous system the chief changes consisted in the involvement of the smaller vessels and precapillaries of spinal cord and of cerebral cortex, as also of the capillaries, in alterations of ganglion cells and in diffuse glia proliferation in various places throughout the cortex. As for the peripheral nerves, acute and chronic changes were visible in the vessels of epineurium and perineurium; no parallelism was discovered between degree of vessel change and of local neural degeneration. In liver, spleen, kidney, pancreas and other abdominal viscera the typical picture of periarteritis nodosa was revealed.

S. A. K. W.

[118] **A clincophysiologic study of the pathway of pain impulses in cardiac disease.**—GEORGE I. SWETLOW. *Amer. Jour. Med. Sci.*, 1929, clxxviii, 345.

As well as recording eight cases of angina pectoris treated by paravertebral injection of alcohol a review on recent work dealing with cardiac pain is included. Operative interference has on the whole proved of little value. Jonnesco failed to bring relief by extirpation of the entire cervical chain along with the first thoracic ganglion; Danielopolu sectioned the cervical sympathetic chain above the stellate ganglion together with the vertebral nerve as well as a nerve which joins the superior cervical ganglion to the cranial nerves; he also removed the cervical sympathetic chain, cut the vertebral nerve, all the branches leaving the vagus to enter the thorax, and the rami communicantes which joined the inferior cervical ganglion and the first thoracic ganglion to the last pair of cervical nerves. None of these operative procedures was entirely successful. Experimentally Jonnesco showed that the cervical sympathetic chain does not convey pain discharges from the heart and aorta. With these facts before him the author examined each of his cases from the neurological standpoint and found that the area of skin supplied by the spinal segments C8 to D6 or D7 was hypersensitive. Accordingly, he suggested that pain impulses from the heart and aorta passed via the posterior roots of the above spinal nerves to the spinothalamic tracts. He then proceeded to inject these roots and ganglia with alcohol and in all cases produced relief from pain for several weeks up to several months. This method appears to be the most hopeful for the cessation of pain in angina pectoris.

E. A. C.

[119] **The Moro embrace reflex in infancy.**—Murray B. Gordon. *Amer. Jour. Dis. Child.*, 1929, xxxviii, 26.

This reflex was first described by Moro in 1918. He found that on placing an infant on a table and then forcibly striking the table on either side of the child, a motor reaction was obtained. The arms were suddenly thrown out in an embrace attitude, describing an arc, and tending to approach one another with a slight tremor; the fingers were at first spread and then closed. Similar movements were observed in the lower extremities. A study of this reflex was made in 300 normal infants and in 21 sick children. It was found to be strongest during the first two months of life, to diminish in intensity from then on and to disappear at the end of the fourth month. Absence on one side suggests either a paralysis or injury, while persistence of the reflex above the age of six months signifies cerebral injury.

E. A. C.

[120] **The early diagnosis of syphilitic pupil changes by means of the pupilloscope of Hess** (Die Frühdiagnose der luetischen Pupillenstörung mit dem Hess'schen Differentialpupilloskop).—S. L. Last. *Deut. Zeits. f. Nervenheilk.*, 1929, cvii, 243.

One hundred and fifty-three normal and 121 patients were submitted to examination with the differential pupilloscope of Hess. Of the 121 patients only 31 gave no evidence of syphilitic infection. Among the syphilitic was a rather large number, diagnosed as merely neurotic or neurasthenic, in whom the pupilloscope proved syphilis to be manifest, and in each instance serum or fluid Wassermann tests confirmed the finding.

A full description is furnished of the employment of the instrument, and its simplicity and quickness of working are stressed as being of direct clinical value.

J. S. P.

[121] **A peculiar form of hereditary familial disease** (Une forme particulière de maladie héréditaire familiale).—Marc Serejski. *L'Encéphale*, 1928, xxiii, 821.

The cases here described are those of three brothers and one sister of the ages of 29, 22, 20 and 18 years who suffered from night-blindness, deafness, mental backwardness, speech defects (dysarthria and agrammatism) and a certain degree of sexual infantilism. Hearing, speech defects and mental retardation had been present since infancy. Night-blindness associated with retinitis pigmentosa came on in all four at the age of 15. The parents were Russians, non-Jewish, and were not related. They showed no stigmata of nervous disease. The author discusses the possible relationship of this condition to the juvenile type of amaurotic family idiocy.

J. G. G.

PROGNOSIS AND TREATMENT.

[122] **Treatment of idiopathic epilepsy by induced malaria.**—J. C. Ramsay. *Jour. Ment. Sci.*, 1929, lxxv, 257.

From observation of six cases, it is not claimed that the results do more than merely point to the possibility of obtaining some degree of benefit. Decrease in the frequency of the attacks may be expected over varying periods. This may be accompanied by some amelioration in the mental state. The reversal of the leucopenia in two cases affords some evidence that desensitization can be attained in patients who show the hæmoclastic crisis. Improvement in symptoms occurs, however, in the absence of this reaction, and there are apparently other factors concerned in bringing about the change. In certain cases the induction of pyrexia is not free from danger.

C. S. R.

[123] **The therapeutic indications and the dangers of the intravenous administration of sodium-phenyl-ethyl barbiturate (sodium luminal) and other barbituric acid derivatives.**—Soma Weiss. *Amer. Jour. Med. Sci.*, 1929, clxxviii, 390.

Soluble preparations of any barbituric acid derivative produce almost instantaneous effect in man when administered intravenously. The symptoms and signs produced are identical with those observed after oral administration. This method of therapeutic application has been used with success to induce sleep in certain psychoses and in eclampsia. It has been found of especial value in status epilepticus. Five cases of tetanus were relieved and relaxation of the muscles took place, rapidly followed by sleep. Persistent hiccough was unbenefited. Sodium luminal used intravenously should be injected in doses of 0·4 to 1·2 g at the rate of 50 mg per minute in a 10 per cent. solution: the dose varied in different cases and should be judged by the immediate effect. The author is careful to point out that such a form of therapy entails potential danger for the patient and should only be used in critical cases after other measures have proved valueless.

E. A. C.

[124] **Researches on a case of castration with transplantation of an ape's testis** (Untersuchungen an einem Kastraten mit transplantiertem Affenhoden).—Josef Wilder. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, cxvii, 472.

This case is of considerable scientific and clinical interest, and is described at great length and in the minutest detail.

The patient was a man of 43, known to be a cryptorchid (one side), who on May 8, 1918, was wounded in the scrotum by a shell splinter. So severe was the injury that eventually a castration was performed, the concealed testis

being also removed. Immediately thereafter sexual desire and power came to an abrupt end, and for eight years subsequently he never had a sexual dream. Full details are given of the physical and psychical eunuchism that gradually established itself, as well as of the definite changes of a neurosympathetic kind that were observed. It is of no little interest that after some time attacks apparently of a narcoleptic kind developed

On December 14, 1928, under anæsthesia, an ape's testis, divided into two, was imbedded under the skin of the abdomen, one piece on either side. One of these was accidently injured and removed, but the other 'took.' Within about three weeks the patient began to lose some of his fat and his hair began to grow again; his abnormal sweating diminished and he became less sleepy. Two months after the operation he had a sexual dream. For the numerous details of the subsequent course of the case (observed for a period of three months), the original deserves perusal.

The conclusion is as follows: "There can be no doubt that as a result of the transplantation a factor was introduced into the body which acted permanently on the vegetative nervous system in the same way as hitherto extracts from the genital organs have acted temporarily, viz., by reduction of the increased vagotonus that followed the castration."

S. A. K. W.

Psychopathology.

PSYCHOLOGY.

[125] **A biochemical approach to the study of personality.**—GILBERT J. RICH. *Jour. Abnorm. Soc. Psychol.*, 1928, xxiii, 158.

BIOCHEMISTRY offers a fruitful means of studying personality, inasmuch as certain chemical determinations appear to be definitely correlated with personality traits.

Preliminary experimentation indicates tendencies for the following relationships to hold:

1. The least excitable individuals tend to have the most acid urine and saliva, while more excitable persons tend toward neutrality or alkalinity of their fluids.

2. The least aggressive subjects appear to excrete the greatest amounts of acid as measured by formol titration and to have the highest alkali reserve of the blood, while more aggressive persons seem to show opposite characteristics.

3. Emotional excitability tends to bear an inverse relationship to the presence of creatinine in the blood and its excretion in the urine.

4. Phosphorus metabolism appears to be bound up with personality in some way, but the details of the relationship are not yet clear.

J. S. P.

[126] **The relation of muscular tonus and the patellar reflex to mental work.**—F. L. Golla and S. Antonovitch. *Jour. Ment. Sci.*, 1929, lxxv, 234.

Though an increase of tonus accompanying mental effort shows a corresponding general increase of the knee-jerk response, isolated knee-jerks may occur during the heightened phase which are not only much smaller than their neighbours, but may actually be much less than those occurring when the tonus is depressed during the resting period. An examination of simultaneous records of the knee-jerk of one limb and the tonic response of the other shows no correlation between the spontaneous variations of knee-jerk and of tonus when the subject is at rest. We may therefore distinguish increases and decreases of knee-jerk depending on mental effort and affective stimuli which are accompanied by parallel variations of tonus; and on the other hand, random individual fluctuations of the knee-jerk not dependent on cerebral activity and not accompanied by any corresponding changes of tonus. There is a large body of evidence which tends to support the view that increased muscular tension improves mental efficiency. It will be found that all this evidence deals entirely with voluntary innervation, and has in itself no direct bearing on the problem of the increases of tonus here dealt with. The authors' experiments lend no support to any attempt to co-ordinate attention with muscular mechanisms. The rise of tonus occurring at the inception of work appears to be of the nature of a preparatory reaction which might fitly be compared with some of the bodily concomitants of emotional states. It is found that the rise of an emotional state is accompanied by certain bodily reactions which do not persist throughout the continuance of the emotion. These reactions are probably an atavistic phenomenon, representing the preparation of the organism for continued bodily activity, the necessity for which no longer exists under modern conditions. Such movements as actually accompany mental processes are of a more purposive type, and belong to another and more recent mechanism than that concerned with emotion or effort.

C. S. R.

[127] **Psychological effects of long spells of repetitive work.**—K. G. Pollock. *Brit. Jour. Psychol.*, 1929, xx, 43.

The following general but tentative conclusions were come to. In all periods of one hour and upwards accuracy tended to decrease in the second as compared with the first half of the spells of work, the relative decrease in accuracy apparently increasing in spells of above one hour, but thereafter remaining fairly constant up to spells of eight hours. There appeared to be a small involuntary increase of rate of work during the second as compared with the first halves of the spells irrespective of their length. In general, accuracy improved slightly after prescribed pauses, more or less irrespective of their length, and seemed to decrease slightly after unexpected pauses enforced by

some disturbing external circumstance. Feelings of tiredness were almost consistently accompanied by a fall in accuracy but by practically no change in the rate of work. Whenever such fluctuating attitudes as cheerfulness and confidence were recorded they seem to mark a period of increased accuracy. Singing invariably appeared to improve accuracy and slightly to decrease rate of work. General drowsiness seemed to produce a slight fall of accuracy and a slight increase of rate. Outside distractions (produced by external continuous noises) seemed slightly to increase accuracy and decrease rate of work. Momentary distractions, such as the coming of individuals into the experimental room, increased accuracy more but had little effect on the rate of work.

C. S. R.

[128] **Results from some new tests of practical abilities.**—W. VICKERS and V. H. HOSKIN. *Brit. Jour. Psychol.*, 1929, xx, 66.

TESTS of practical abilities have been devised giving consistent results. Practical ability increases up to the age of 25 to 30 and then decreases, whereas practical skill (at least in file-cutting and machine tool making) increases progressively up to 50 years of age. Intellectual ability, when tested in a practical situation, also increases to a maximum at 25 years and then decreases. Age is thus shown to act as an interference factor. The women scored less than the men in all the tests.

C. S. R.

[129] **Some notes on the attitude of children to dress.**—EVE MACAULAY. *Brit. Jour. Med. Psychol.*, 1929, ix, 150.

CHILDREN up to about the age of nine look upon clothing simply as being decorative, and would probably, if free to choose, wear anything as long as the colour is sufficiently brilliant. Clothing has little, if any, significance for the self and is not considered when the claims of the body for physical enjoyment are to the fore. From the tenth to the twelfth year the desire for decoration increases, and some slight attention is given to the actual design and cut as well as to the colour. The edicts of convention also oppress the child during this period, and there is some conflict between the desire of the body for unrestricted enjoyment and the necessity for caring for and considering the clothes. During adolescence begins the sublimation of interest from the body to the clothes, probably accompanied, on the part of girls, with a certain amount of consciousness of sex. The motive of modesty is strongly considered in connection with clothes so far as the females in the lower sections of society are concerned. Those at the bottom of the social scale have been unwilling to accept the now widely held opinion that a lightly veiled body is not necessarily and deliberately immodest. Probably the unconscious impulse, up to the ninth or tenth year, is in favour of nakedness.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[130] **Compulsive thinking as a castration equivalent.**—E. Graham Howe. *Brit. Jour. Med. Psychol.*, 1929, ix, 159.

There is a group of cases, varying between particular obsessional ideas and general obsessional thinking, in which the act of thinking has become compulsive in motive, excessive in degree and exaggerated in value. These are here alluded to as "compulsive thinkers." The underlying unconscious motive is escape from the conflict of an unsolved emotional problem, which is due to a greater or less degree of parent fixation. The escape is by way of a "castration" or de-emotionalization process, in which genital values become transferred to thinking processes in general. The symptom-complex of the compulsive thinker is suggestive of the dementia præcox type, viz. an escape from reality into thinking of a compulsive character, emotional regression and sexual maladjustment, isolation, martyrdom; self-punishment, castration symbolism and suicidal tendencies. The "inviolable personality" is a marked feature of the compulsive thinker and is due to the transference of genital value to the thinking process. Psychotherapy is peculiarly difficult for various reasons. The critical faculty is itself involved in the compulsion. The inviolable personality must be violated, and when threatened always tends to revert again to defend itself by compulsive thinking. Fear of emotional reality is so strong as to cause fear of recovery, and sanity to be feared as being itself insane. The subtlety and widespread character of the process make analysis especially difficult. Compulsive thinking is more than a problem in individual psychopathology, for its principle permeates the foundations of society.

C. S. R.

[131] **Dominating, over-emphasised, compulsive and delusional ideas** (Dominierende Vorstellung, überwertige Idee, Zwangsvorstellung, Wahnidee). —D. Adam. *Allg. Zeits. f. Psychiat.*, 1928, lxxxix, 383.

Dominating ideas come within the experience of everybody, and are quite normal. A train of thought interrupted or a solution delayed by external circumstances, if associated with great emotional interest, dominates the mind and though recognised fully as of subjective origin is felt as annoying or distressing by reason of the tension it produces, until a solution is reached and it becomes disposed of among the ordinary contents of the mind. It is not, even when dominant, felt as a foreign body imposed on the mind from without.

The over-emphasised idea is more objective, though not necessarily pathological, and is not felt as distressing in its dominance. Such exaggerated ideas backed by strong emotions seem to become as it were imposed on the owner and guide his actions; an example given is the case of a man exaggerating a trifling injury and spending all his energies for years in striving to obtain a pension,

or that of individuals harassed by a sense of inferiority. The excessive emotional tone of the dominating idea and its acceptance as the chief interest in life are the main characteristics.

Compulsive or obsessional ideas are recognised by those who have them as being strange and unusual, logically false and unreal, yet possessing a sort of compelling belief in them ; the intelligence is unimpaired, and they lack the strong emotional tone of the over-emphasised ideas. Obsessing thoughts, tunes, fears and actions are sub-headings. The sufferer is well aware that the obsession is unreasonable, but feels obliged to act in accordance with it. This distinguishes it from the delusional idea.

Delusions are usually accompanied by strong emotion and represent emotional relationships with the environment. They may be described as falling into two main groups : those representing wishes and hopes of the patient such as grandiose delusions, and those whose content is his fears, as in delusions of jealousy, persecution, hypochondria, and of wickedness or unworthiness. They may arise on a basis of an over-emphasised idea which the patient has failed to conquer or resolve into his general personality, so that he becomes incapable of correcting its falseness.

Conclusions : the dominating idea is subjectively compelling and therefore felt as a painful feeling of lack of freedom of thought.

The over-emphasised idea is objectively compelling, the bearer identifying himself with it, and therefore without influence on feeling. Both of these arise on a basis of exaggerated feeling-tone, and cannot be spontaneously shut off ; both may have a normal content.

Obsessional ideas are subjectively compulsive, sometimes without affective basis, not necessarily accompanied by emotion or incapable of being shut off ; they are pathological in their content and subjectively recognised as abnormal, and therefore rejected by reason.

Delusions are objectively compulsive, distorted in content, arise from morbid causes and are incapable of correction.

M. R. B.

PSYCHOSES.

[132] **Basal metabolism in schizophrenia.**—R. G. Hoskins and F. H. Sleeper. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 887.

Determinations of the basal metabolic rate in persons with dementia præcox showed that the rate averages more than 10 per cent. below normal.

R. M. S.

[133] **Millon's reaction in the urine in toxic psychoses** (La reazione di Millon dell'urina nelle psicosi tossiche).—E. Scheiner. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 298.

The author maintains that in alcoholic psychoses and in the amentia syndromes

of postinfective, postpuerperal and similar psychoses, and in hebephrenic-catatonic dementia præcox, the urine constantly shows a substance positive to Millon's reaction. This is not free phenol or an aromatic acid or tyrosin. Accompanying the substance, which seems to be due to a destruction of liver cells, are other substances characteristic of hepatic degeneration such as urobilinogen and urobilin.

R. G. G.

PSYCHOPATHOLOGY.

[134] **The relation of unresolved infective processes following acute infective diseases to the causation of mental disorders.**—T. C. GRAVES. *Jour. Ment. Sci.*, 1929, lxxv, 31.

CHRONIC infective processes in cases of mental disorder are generally of long standing. They do not merely include focal infections in the form of diseased teeth, tonsils, nasal sinuses, gall-bladder, etc. Such a process implies a general biochemical disorder, the full extent of which may not be fully realised, and of which one example may be a calcium deficiency. It has generally been preceded by an acute infective process more or less well marked, which subsides but leaves behind areas of unresolved infection, and in these areas general poisoning goes on. Here are emphasized, and illustrated by specific cases, mental disorders associated with unresolved infection such as that following measles, scarlet fever, encephalitis lethargica, pneumococcal, intestinal, influenzal and other septic infections, sometimes pure and in other cases mixed. Why the acute infective process associated with these diseases should leave behind it areas of unresolved infection the author does not know, but supposes that there may be some local maldevelopment of structure together with a general biochemical disorder. As regards influenza and mental disorder some interesting conclusions have been reached. (1) In persons without psychotic inheritance but with pre-existing septic states in the head, an attack of influenza by causing an acute exacerbation of the old pathological process may precipitate serious mental disturbance, even though constitutional symptoms of influenza may be slight or absent. (2) The mental symptoms and the corresponding pathological conditions of septic foci with deficient or defective drainage may continue indefinitely. (3) The usual influenzal symptoms displayed by a relatively healthy person may show considerable differences from those occurring in persons with an existing septic process in the head. (4) Within the skull, therefore, pathological processes may exist, the extent of which may determine the degree of mental symptoms displayed.

The cases of encephalitis lethargica investigated have fallen into two main types—the chronic and the acute. The former were cases in which the patients had passed into a chronic stage, and with progressive mental and physical deterioration became certified after many years. In these cases areas of unresolved infection had been operating over a long period of time. On admission,

the acute cases showed acute mental symptoms with definite evidence of ocular palsies, ptosis, pupil disturbances, Parkinsonism, etc. In some, treatment of gross dental sepsis was followed by mental and physical improvement, by recovery and satisfactory reports subsequently. In others, chiefly adolescents, septic tonsils, adenoids and infected nasal sinuses were found. On removal or drainage mental and physical improvement and discharge followed. In these there was definite pathological conduct and in the females menstrual irregularities. Following the removal of the septic disease in the head menstruation became regular and conduct normal.

C. S. R.

[135] **The relation of focal infection to mental disease.**—Nicholas Kopeloff and George H. Kirby. *Jour. Ment. Sci.*, 1929, lxxv, 267.

The authors' published studies on the subject led to the conclusion that focal infection could not be regarded as the essential etiological factor in the functional psychoses, and that surgical detoxication resulted in no greater a number of improvements or recoveries than occurred in patients not operated on. The adverse criticisms of Hunter are discussed and refuted. There are three important questions requiring consideration in dealing with the relation of local infection to mental disease :

1. Is focal infection the specific *cause* of the functional psychosis ?
2. Can the surgical removal of foci of infection *alone* bring about improvement and recovery of patients with mental disease ?
3. Is focal infection of importance in mental disease ?

Kopeloff and Kirby's studies indicate clearly that the first two questions must be answered in the negative. No essential differences were noted between the group receiving the usual hospital treatment and the members of another group receiving surgical treatment to remove the foci of infection. In a word, operative procedure *alone* was not responsible for mental improvement or recovery. With regard to the third question, it can only be said that so long as there exist no satisfactory criteria for evaluating the relative importance of each of the factors which enter into mental disease we must suspend judgment and gather further facts or develop better methods.

C. S. R.

[136] **Gastro-intestinal focal infection in relation to oral sepsis, with special reference to anærobes, occurring in six cases of mental disorder.**—W. M. Ford Robertson. *Proc. Roy. Soc. Med.*, 1929, xxii, 1373.

From the consideration of 114 cases it is concluded that in mental disorders gastric dysfunction, especially hyperchlorhydria, is present to a much greater extent than in normal individuals. The importance of free hydrochloric acid as a bactericidal barrier has been proved and the relationship between this fact and that of the production of gastritis has been established. Adequate

bacteriological methods show that oral sepsis can be traced throughout the alimentary canal. The degree of gastric infection does not increase the expectation of invasion of the intestine. Anærobic methods are essential if the full significance of the special types of infections present in mental disorders is to be realized.

C. S. R.

[137] **Mental deficiency and maladjustment.**—HENRY HARRIS. *Brit. Jour. Med. Psychol.*, 1928, viii, 285.

MENTAL deficiency is a maladjustment due primarily to biological defect causing mental subnormality, due secondarily to psychological or social factors or both. Where adult intelligence does not exceed a Terman age of six or seven—as in the idiots and imbeciles—the primary biological factors in themselves constitute mental deficiency. Where the Terman age lies approximately between seven and eight or nine, it is the presence of the secondary factors that determines whether a given individual will ultimately be classifiable as legally defective or as merely subnormal.

The primary biological factors determining subnormal intelligence are manifold; mental defect is not a unitary condition. Of the five groups—hereditary, blastophoric, congenital, natal, and acquired—the second and third are especially worthy of further investigation by geneticists, obstetricians and others. Hereditary factors will demand a eugenic solution; blastophoric and somatic factors a euthenic solution. At the moment it seems wiser to consider both methods of attack, with the emphasis perhaps on the latter.

Mental defect is most practically considered in terms of intelligence and social behaviour.

The psychological factors complicating subnormality consist, for the most part, of anomalies of temperament and character: to a lesser extent of psychotic and psychoneurotic reactions. At the moment, the most practical method of rating temperament is to attempt an evaluation of emotional traits that have been selected on a purely situational basis. Anomalies of temperament and character are then the exaggeration of individual emotional traits or of a disharmony between them, and are the result of social factors in the environment interacting on the increased suggestibility of the subnormal. Failing prevention by general social and educational measures, such anomalies will require individual corrective treatment by parent, teacher, probation officer, or in the last instance by the psychiatrist aided by his social workers.

Social methods of treating and controlling mental deficiency have the following aims. The biological determinants of subnormal intelligence must be kept in check by positive and negative, eugenic and euthenic, measures. The subnormal must be socialized to the full extent that his intelligence allows. A third aim is to control the environment, and to avoid features which tend to induce character anomalies or precipitate antisocial reactions. Fourthly,

custodial and antisocial types must be segregated. Every effort must be made to socialize as many as possible of the latter by establishing a systematic, continuous, progressive chain of contacts with the outside world.

AUTHOR'S ABSTRACT.

[138] **The Arneth blood-count in idiocy and low-grade imbecility, with special reference to the incidence of tuberculosis and tubercular infections.**—STANLEY R. TATTERSALL and CYRIL J. THOMAS. *Jour. Ment. Sci.*, 1929, lxxv, 64.

THE technique of the Arneth blood-count is explained. From their work the writers conclude that this procedure has proved itself to be an aid to diagnosis in many cases. In several cases where physical examination has been either impossible or unsatisfactory, a low Arneth count has rendered probable a diagnosis of tuberculosis before physical signs have been discoverable by other means. To those engaged in the work of a mental hospital, any method of assessing the progress of a tuberculous patient is valuable. Its value is the more apparent when the measure is easy of application. The Arneth count enables the physician to segregate at an early date his infected patients, and to lessen the incidence of a disease which finds a favourable soil in the mentally defective population.

C. S. R.

[139] **Congenital wordblindness and agraphia in a feebleminded subject** (Un cas d'aléxie et d'agraphie congénitale chez un débile mental).—O. DECROLY. *Jour. de Neurol. et de Psychiat.*, 1929, xxix, 346.

A DETAILED study of a young boy of defective intellect, whose major disability concerned the faculties dependent upon vision, especially reading and writing. He is not to be included among the cases of pure congenital word-blindness of Hinshelwood on account of the co-existence of other intellectual defects. Although the author does not anticipate a complete development of the ability to read and write he has noticed a definite improvement in this direction as the result of careful education.

M. C.

[140] **Alzheimer's Disease.**—W. MALAMUD and K. LOWENBERG. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 805.

THE author discusses the clinical course and pathological observations in two cases, both of which showed the changes found in Alzheimer's disease: they differed, however, in that one occurred at the usual age, whereas the other occurred early in life.

Case 1. A man, in whose heredity there was a tendency to senile psychosis and whose history previous to the onset was essentially unimportant, began to

show signs of mental disturbance at the age of 62. These consisted at first of spells of dizziness, defects of memory and disturbances of speech. Following an apparently complete remission, the symptoms recurred for another short period, to be followed by a less complete remission of a few months. This, in turn, was followed by an interrupted progression of the disturbance of memory and mild paranoid ideas. On admission (fifteen days before death) he was semistuporous, and showed only slight indication of paresis on the right side with questionable pyramidal tract signs. There were disturbances of speech in the form of aphasia and paraphasia, as well as a tendency to logoclonia. There was definite apraxia, difficulty in obeying commands other than of the simplest type, and disturbances of writing and copying. He was restless, impulsive and untidy in his habits; the Wassermann reactions of the blood and cerebrospinal fluid were negative. The fluid did not show any pathological changes other than slightly increased permeability. Postmortem examination showed a carcinoma of the cæcum and mild arteriosclerosis. The brain did not show gross changes, but histologically it revealed the following outstanding features: numerous plaques and Alzheimer cell changes throughout the cortex, with the exception of the occipital pole, which did not show the latter; degenerative phenomena in the ganglion cells and neuroglia, especially marked in the basal ganglia, and mild degenerative phenomena in the vessels of the cortex and the pia, but no signs of arteriosclerosis or inflammation. The choroid plexus showed changes that at the age of the patient are to be considered mild and non-specific. The presence of rich deposits of iron completes the picture of a pronounced degenerative disease of the brain.

Case 2. Following an infectious disease at the age of seven, the patient, who is said to have been normal before, showed a gradually progressing mental disorder expressed in inefficiency in school work and social tendencies. This chronic picture led to periods of excitement, restlessness and impulsiveness, which necessitated confinement. A further mental decline took place, with acute exacerbations alternating with periods of quiescence. Following a period of intense disturbance, characterised by muscular tension, mutism alternating with rambling speech, impulsiveness, extreme untidiness, confusion, apprehensiveness and so forth, there was another period of quiescence during which the patient was taken home, where he remained for more than four years. During this period he is said to have been fairly tractable although needing somewhat constant supervision. This was followed by a final exacerbation which began about six months before death, apparently associated with a slight respiratory infection and with mental symptoms that were like those of previous occasions. They were characterised especially by extreme restlessness, confusion, untidiness, wandering at night and disturbances of speech. Death occurred from exacerbation of an old tuberculosis process. At postmortem examination there was found an acute tuberculous process, but not any other gross lesions. The brain showed histologically: (1) an

extremely advanced, typical picture of Alzheimer's disease, with numerous plaques, Alzheimer cell changes and fibril degenerations, and associated with it a marked nonspecific degenerative process throughout the cerebral cortex and basal ganglia, the cerebellum being intact: (2) a condition of the choroid plexus consisting of an extensive degenerative process of the vessels of the villi. The lumen in most of these was completely obliterated by concentrically arranged rings of structureless substance in the walls. Some of the vessels, however, were still patent and contained blood. The connective tissue surrounding the blood vessels (especially the capillaries) was greatly increased and showed signs of an advanced degenerative process. The epithelial layer was represented by a thin margin on the periphery; the individual cells were degenerated.'

According to the authors we must discard the notion of Alzheimer's symptom-complex being particularly bound to a certain age, and from a pragmatic point of view there is not any particular advantage in regarding the whole group as an expression of presenile changes. Etiologically and pathogenetically we know little about the process of senility other than that it may be produced by some unknown metabolic changes and the possibility of a disturbance of the choroid plexus producing a picture of this type in one case offers a stepping stone toward discovering such a common factor. The results of recent investigations into the physiology of the plexus, and the possible rôle it plays in the exchange of substances between the blood and the cerebrospinal fluid, give valuable clues as to the direction to be followed. The changes which take place with age, even normally, may be of further assistance.

R. M. S.

[141] **The diagnosis of general paralysis of the insane as a clinical and pathological entity.**—JOHN BRANDER. *Jour. Ment. Sci.*, 1928, lxxiv, 673.

AFTER some historical survey, it is pointed out that the diagnostic criteria of general paralysis and its fatal prognosis were developed many years before the days of Argyll Robertson and Wasserman. Many cases now included under this heading would not formerly have been, because they did not resemble general paralysis. The introduction of the Wasserman reaction has certainly helped to eliminate some doubtful cases but has brought fresh errors in its train. It will probably be conceded that complete recovery from the signs and symptoms of cerebral syphilis means meningovascular disease. Of those conditions which resist treatment, some are due to parenchymatous syphilis, and some are the result of irreparable destructive lesions caused by processes in the vessels or meninges. There is a tendency to accept certain isolated phenomena as unequivocal evidence of parenchymatous syphilis, viz., the Argyll Robertson pupil, loss of the knee- and ankle-jerks, and primary optic atrophy. These, however, may occur as end-results of ordinary meningovascular disease.

Can we discriminate between these two conditions by means of the Wasserman reaction? The writer thinks not, in the present state of our knowledge. Reference is made to certain differences in the pathological findings of typical general paralysis and the mixed types. There is some evidence that tertiary syphilis does not occur in association with general paralysis: yet the so-called mixed forms of general paralysis are avowedly cases of combined tertiary syphilis and parasyphilis. Cases are here quoted which are believed to constitute a considerable class and which are able to affect appreciably the statistics of general paralysis if by chance they are confused with this disease. Cerebral syphilis assumes so many different forms and operates in so many different ways that no unusual clinical finding should be given a final interpretation until after a post-mortem examination. It is of importance that a definite distinction be maintained between typical general paralysis and the mixed forms. Such distinction has a practical bearing on the study of causes, the interpretation of therapeutic results and the development of fresh methods of prevention and cure.

C. S. R.

[142] **The hæmatopoietic functions in general paresis.**—H. WILFRED EDDISON. *Jour. Ment. Sci.*, 1929, lxxv, 242.

IN general paresis there is impairment of the function of the leucoblastic tissues in the bone-marrow, with diminution of the leucocyte reserve. This is not the case in non-paretic syphilis. General paresis is differentiated from other forms of cerebral syphilis by the nature of the parenchymatous changes in the central nervous system. The affection of the blood-forming tissues constitutes another differentiating factor. It is possible that this condition is one which depresses the cerebral tissues and weakens their resistance to the attack of the spirochæte. An analogy can be drawn between paresis and pernicious anæmia, in that (1) there is, in both, impairment of the blood-forming tissues; and (2) the distribution of the degenerated areas in the medulla and spinal cord is suggestively similar in both. The depression of leucopoietic activity may account for the ease with which bed-sores are developed in paretic subjects, and the readiness with which the patients die of intercurrent infection. When some infection is induced in a paretic which will excite polymorphocytosis, the circulation immediately receives a high proportion of immature leucocytes. The degree of leucocytic reaction induced by infection with *Spirochæta Obermeieri* or other leucogenic agents varies inversely with the stage to which the general paresis has advanced. The better the general condition of the patient after such pyrexial treatment the nearer does the differential count approach the normal, and the converse is equally true. The progress of general paresis can sometimes be stayed by measures which result in increased leucogenesis. On the other hand, these measures tend to hasten the progress of the disease in cases which produce

an unfavourable leucocytic response. With regard to induced malaria, which, so far as the polymorphocytes are concerned, produces leucopenia, it is significant that a condition which gives the leucopoietic tissues a rest should be associated with a clinical improvement. It is suggested that when pyrexial therapy of a leucogenic type is contemplated, a preliminary test should be carried out in order to eliminate those cases which give a poor leucocytic response, since these are apt to deteriorate under such treatment.

C. S. R.

[143] **Medical examination prior to marriage and mental disorders** (Examen médical prénuptial et troubles mentaux).—L. REDALIÉ. *L'Hygiène mentale*, 1929, xxiv, 15.

AFTER discussing the various points connected with such an examination, the writer concludes that in the present state of knowledge any medical certificate would not solve such a complicated problem as that of pathological character or psychopathic heredity in their relations to marriage. The obligation of having such a certificate risks an increase in the number of free unions and thus of not preventing the procreation of degenerated stock. The best means to combat undesirable marriages from the point of view of medicine consists in the propagation of mental hygienic ideas.

C. S. R.

PROGNOSIS AND TREATMENT.

[144] **The psychology of readjustment, with special reference to mental hygiene work in college.**—KARL F. MUENZINGER and FLORENCE WEAVER MUENZINGER. *Mental Hygiene*, 1929, xiii, 250.

IN the process of readjustment, according to these writers, the factors to be considered are: clarification and change of purpose, counterbalancing thoughts, substitution of overt responses ('habits'), attaching emotional reactions to new persons and situations, appeals, counteraction by fear of consequences, the attitude of detachment, definiteness of attack and planning ahead, self-help, and change of environment. The last named is often difficult, if not impossible, to effect. If a change of environment is not planned, the student realizes from the beginning that there is no possibility of 'running away' from his difficulties. He must face these frankly and solve his conflicts rationally. This means an insurance against future maladjustments. This change of the psychological situation at times means the substitution of conscious desires and purposes for unconscious ones. In guiding a change of the psychological situation of the maladjusted, the adviser cannot hide entirely his own personal bias and prejudice. He must always be aware of the necessity of preserving the integrity of the maladjusted person and not inject his own desires. It may even be necessary that the adviser warn the student not to

accept the adviser's evaluation of the factors of the situation but to work out his own. Such dilemmas have been found especially in difficulties of a sexual or religious nature.

C. S. R.

[145] **Child-study, the modern hobby.**—MARY CHADWICK. *Psyche*, 1929, ix, 69.

IF some parents treat the education of their children as a hobby so that they may be induced to experiment too much or never allow the child enough elbowroom to develop along its own lines, but only in those strictly laid down by authorities, another type of modern parent runs to the opposite extreme of repudiating the responsibilities of parenthood and in the guise of giving the child unlimited freedom, withdraws the help and support that the child needs and expects from those older than itself. Unconsciously it is a revenge reaction, and a coward's method of escaping from any blame that may arise afterwards. Whatever evil consequences make havoc of the children's future lives, these parents feel they will not be censured—for they will claim they left the children to do as they liked, to take their own way and to learn through experience. This seems one of the most direct ways, although it may be carefully disguised, of exposing the infant so that it will be destroyed and not become a future rival to the parent's prosperity. Unconsciously the parent does nothing because he or she fears reaction. He fears blame and therefore remains inactive.

C. S. R.

[146] **Old and new methods of treatment in general paralysis : a comparison of results.**—G. E. SHAND. *Jour. Ment. Sci.*, 1929, lxxv, 250.

THE results of treatment by the following means were compared—Ford Robertson serum : urotropin : sodium nucleinate : and salvarsan, mercury and potassium iodide. Out of 12 serum cases, two showed marked improvement. In seven others there was considerable improvement, but none were considered fit for discharge, and in eight to ten months' time they again deteriorated. In three cases there was no improvement, and two of the patients died from convulsions after the treatment had stopped. Four cases were treated with urotropin, grs. 15, thrice daily. Only one of the four showed any improvement. He was discharged to his friends, but soon afterwards had to be sent to another hospital. With sodium nucleinate none of the patients showed the slightest improvement. The other cases were given 0.6 g salvarsan intramuscularly, three doses at weekly intervals. This was followed by mercurial inunction and medium doses of liq. hyd. perchlor. and potassium iodide, continued for twelve months. There was no change in the mental condition, and both patients died shortly after from convulsions. The four control cases were interesting in that three of them showed remarkable

improvement. Two patients, in fact, were discharged to the care of friends, but one returned in a fortnight and the other in a couple of months and died within a month or two of their return.

C. S. R.

[147] **The enigma of progressive paralysis** (L'enigma della paralisi progressiva).—M. ZELLA. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 352.

THE author examines the question of the influence of vaccination and smallpox on the incidence and progress of general paralysis, but concludes that there is no relationship between the two. He also concludes that whatever the therapeutic value of malaria it has no preventive influence.

R. G. G.

Reviews and Notices of Books.

Mental Deficiency (Amentia). By A. F. Tredgold, M.D., F.R.C.P., Lecturer on Mental Deficiency, London University, etc. Fifth edition, largely rewritten. With 35 plates. London: Baillière Tindall & Cox. 1929. Pp. 535. Price 25s.

The fifth edition of Dr. A. F. Tredgold's book on mental deficiency appears opportunely. The increasing attention paid to problems of the mental defective by legislators, sociologists, psychologists, and others outside the ranks of the medical profession has not always been accompanied by due appreciation of their fundamental basis in an imperfectly and unequally developed nervous system. Even if we prefer Sir Oliver Lodge's dictum "no neurone, no manifestation of mind" to that of "no neurone, no mind," to which latter statement we believe most neurologists at least will subscribe, we should still be compelled to study the imperfect instrument, and the anatomo-physiological substratum of its incompleteness, if we are to gain any insight into conduct disorders and asocial outbreaks. Dr. Tredgold's position is clearly set forth in a single sentence: "Whatever may be the relation of mind to brain, it is now fully recognised that the manifestation of mental activity is indissolubly connected with the cells of the cerebral cortex." Dementia follows their degeneration and death, while amentia, or mental deficiency, is associated with their incomplete development. Obviously, then, the radical problem of the mental defective resides in discovery of the antenatal, natal, and post-natal physical events calculated to injure or retard the growth of cortical ganglion cells. This is a problem in neurology and not in psychology. If prevention is better than cure, and early etiological recognition and possible treatment preferable to years of segregation and hospitalization, then any increment of knowledge in regard to germinal factors, brain infections, endocrine disturbances, and so forth, is bound in the end to simplify the complexities of the question. To this desirable end Dr. Tredgold has materially contributed by his remarkably full and detailed descriptions, embodying the most recent advances in knowledge.

It may doubtless be claimed that the subject of the moral delinquent or imbecile to which Dr. Tredgold assigns an especially interesting section of his book, forms a problem in psychology. But the question at once arises whether such moral delinquency is not ipso facto a sign of intellectual defect. Social misconduct, in one of Dr. Tredgold's own instances, developed where there was "no obvious intellectual defect," yet the admission of "pronounced

deficiency of wisdom " is made. In our view this cannot be interpreted as other than a defect of mind, which must we maintain be based on cortical misendowment or imperfection of evolution.

With numerous practical questions of education, training, treatment, with those of a sociological and legal nature, this new edition deals in extenso, providing therefore a kind of encyclopædia of mental deficiency of outstanding serviceableness.

S. A. K. W.

Sleep and the Treatment of its Disorders. By R. D. GILLESPIE, M.D., Physician and Lecturer in Psychological Medicine, Guy's Hospital, etc. London: Baillière Tindall & Cox. 1929. Pp. 267. Price 7s. 6d. net.

DR. R. D. GILLESPIE has written a fine little volume on sleep and its manifold problems, which we cordially commend to the busy practitioner and to the expert in nervous or mental disease as well. For here, couched in a pleasantly readable style, will be found a more or less complete exposé of present-day knowledge in respect of the physiology of sleep mechanisms, the pharmacology and mode of action of sedative medicaments, and a highly practical description of how and when to treat any given variety of insomnia. Dr. Gillespie is conversant with the numerous theories of sleep that have at one time or another gained favour, and is sound in his criticism and adequate in his reasons for selection from among them of those on which some reliance may be placed. It is eminently satisfactory to note his conclusion that " the evidence points to the basal grey matter as being a waking-centre and not a sleep-centre," seeing that endless confusion has arisen from the failure of many writers to observe the simple rule that destructive lesions destroy function; if a patient sleeps all the deeper because of destruction of his regio hypothalamica by a neoplasm that area cannot be regarded as constituting a sleep-producing centre, but precisely the reverse.

The reader cannot fail to be impressed by the combination of scientific erudition and skill in a complicated art revealed in these pages, though he may gently remonstrate with their author for providing only a selection, in the bibliographical tables, of the abundant references scattered through the text.

Die Optische Allæsthesie: Studien zur Psychopathologie der Raumbildung. By Dr. GEORG HERRMANN and Dr. OTTO POTZL, Prag. With 17 illustrations. Berlin: S. Karger. 1928. Pp. 302. Price Mk. 28.

THE term allæsthesia is as far as the tactile field is concerned synonymous with allochiria and signifies reference of the local signs of cutaneous stimulation to the opposite side of the body from that to which in reality they belong. This tactile allæsthesia is known occasionally in tabes, Brown-Séquard lesions, and other spinal and root affections, and has been described also in some cerebral cases. By optic allæsthesia the authors mean the somewhat more

rare condition in which seen objects are misplaced in the visual field, being referred to a site different from their actual one (right instead of left, above instead of below, etc.). Apparently the first recorded instance of this anomaly is that of Beyer (1895), who suffered himself from migraine and who made the curious observation that during the occurrence of the migrainous visual phenomena objects situated in reality in the left peripheral visual field seemed to have their locus on the right, and higher up. Subsequent allusions in a sparse literature are summarised by the authors, who then furnish a long analysis of several personal cases of the same class, forming indeed in one or two instances an illustration of mirage effects—a "fata morgana of the visual field," as it has been termed.

As a result of their investigations, which have been conducted in a careful and elaborate fashion, the authors conclude that two factors contribute to the development of this visual allæsthesia, viz., (1) a disorder interfering in part or in whole with the functions of central vision, and (2) a release or escape of function of certain components out of the total influence exercised by the occipital convexity on the area striata. Thus in one case exhibiting loss of central vision, preservation of the peripheral field, and a confusion of left with right in visual space, two lesions were discovered, one (an old one) implicating the left occipital pole, and the other (recent and progressive) starting from the right occipital convexity and spreading towards the calcarine region. This monograph raises many questions of a difficult and rather technical kind which are nevertheless of much interest to the scientific neurologist, and merits reading in extenso by all to whom the subject appeals.

S. A. K. W.

Nervensystem und Spontane Blutungen. By Dr. RUDOLF SCHINDLER, Munich. With five illustrations. Berlin: S. Karger. 1928. Pp. 68. Price Mk. 4.20.

THE occurrence of spontaneous hæmorrhagic effusions in various nervous affections, organic or otherwise, deserves close attention. In respect of hysteria and allied psychoneurotic conditions a wave of scepticism seems to have succeeded the supposedly somewhat uncritical acceptance of the genuineness of the phenomena by older writers, and the present-day attitude is rather one which attributes them to artefact. The inaccuracy and unjustifiability of this way of considering them are strongly animadverted on by Dr. Schindler, who believes such spontaneous bleeding to be much more common than is usually thought, and cites various personal cases (both male and female) proving the contrary. Some of these, indeed, are distinctly impressive, as are also his selected quotations from the large literature on the subject. He provides the reader with an interesting excursus on the question of hysterical stigmata mimicking the wounds of the Christus.

According to his researches, the following conditions should be distinguished: hæmophilia; senile purpura (traumatic in origin); avitaminoses (scurvy, Barlow's disease); blood diseases; cholæmia; toxicoses and infections (hæmorrhages in typhus, smallpox, malaria, scarlatina, syphilis, uræmia, benzol, salvarsan, and many more); thrombopenia. There still remains the wide and controversial group of hæmorrhagic diatheses in association with neurotic and psychoneurotic states, a group the authenticity of which can no longer be denied, and he concludes that they are insufficiently explained by vasomotor dysfunction, for they arise in differing fashions and affect any and every conceivable part of the neuronic system. Relying on the physiological occurrence of spontaneous bleeding in the female sex (menstruation), and its admittedly common alteration by psychogenic agencies, Dr. Schindler argues that too much attention has hitherto been devoted to blood chemistry and vessel wall, and too little to the possibility of central changes in the sympathetic ganglia and corresponding cerebral (hypothalamic) centres. Allusion is made to the recent work of Castex, who has found profound alterations in the upper sympathetic ganglia in two cases of symmetrical purpura in arteriopathic subjects.

Die Paroxysmale Lähmung. By Dr. Otakar Janota and Dr. Klement Weber, Assistants in the Psychiatric Clinic, Prag. With 7 illustrations. Berlin: S. Karger. 1928. Pp. 118. Price Mk. 11.

Periodic family paralysis is a rare condition, opportunities for studying which do not often arise. The present monograph is based on the clinical examination of four cases, coming from three families, and provides an excellent summary of all that is known on the subject, with references to the literature since 1918. As the pathology of the affection is practically unknown theories of pathogenesis, diverse and conflicting, flourish. According to the authors it is possible to exclude all sources for the motor phenomena which are situated central to the motor nerve end-plates: their own view is that the disorder resides in the muscle cells, in that part of the function of muscle innervation which is dependent on the vegetative component (as opposed to the oxydative-chemical component) in muscle tissue. Yet there are certain clinical features of periodic paralysis to which they might have given rather more consideration, and which are a little difficult to explain on the view that it is a variety of 'myopathy,' viz., its occasional hemiplegic type, its association with epilepsy, and, indeed, their own discovery that during the attack the electrocardiogram of the patient exhibits certain definite abnormalities. As far as we have noticed no allusion is made to the phenomena of cataplexy, which in a sense represents an acute and extremely brief attack of myoplegia, characterised none the less by atony, immobility, and loss of tendon reflexes.

The problem of this disease-condition is peculiarly involved, and the monograph under review clearly states the limits of present-day knowledge in regard to it.

L'Epilépsie ; conceptions actuelles sur sa pathogénie et son traitement. By Dr. PH. PAGNIEZ. Paris : Masson et Cie, 1929. Pp. 199. Price fr. 26.

THE researches of the past thirty years on the pathogenesis of the epileptic states and in particular the employment of more accurate biochemical standards have resulted in an accumulated mass of data which is difficult to sift. Herein lies the value of such a book as the present, which strives to evaluate the evidence collected, to separate the important from the unimportant, and to review the entire situation in the light of modern conceptions. We note a gradual shift from clinical and experimental methods to those of the biochemical laboratory and there is a real danger that attention may be diverted too far into metabolic bypaths without material increase in our knowledge of the epilepsies. Dr. Pagniez enumerates and examines the new matter : as might be expected, however, his conclusions are purely tentative, and we do not see any immediate prospect of solution of the old problems—the neurigenic as opposed to humoral conceptions : the priority of vascular over neuronic discharge : the rigid separation of cause from effect—in short, the vexed problems of last century remain as intangible as ever. This work is to be recommended, however, as a balanced and useful presentation of the subject, and it contains an adequate bibliography.

M. C.

An Outline of Neurology and its Outlook. By Sir E. FARQUHAR BUZZARD. London : Oxford University Press, 1929. Pp. 24. Price 1s.

THIS little pamphlet represents the text of the eleventh Earl Grey Memorial Lecture delivered by Sir Farquhar Buzzard at Armstrong College, Newcastle-on-Tyne. The address, which was read to a lay audience, gives a clear and interesting account of the development of neurology as a branch of philosophy, and an able exposé of the present scope of the subject. In an easy and scholarly manner the author describes how our knowledge of the science has developed along five distinct avenues, viz., those of anatomy, physiology, pathology, psychology and clinical observation.

Occasional Notes.

INTERNATIONAL NEUROLOGICAL CONGRESS, BERNE, 1931.

At the instigation mainly of Dr. B. Sachs, of New York, who is well-known as one of the senior members of the neurological profession in the United States, a Committee representing the American Neurological Association sent out circulars to the chief countries of the world suggesting that an International Neurological Congress might with advantage to all concerned be called for 1931, and inviting consideration of the project. To this preliminary communication numerous responses were obtained, indicating unanimous approval of the plan. In accordance with a suggestion made by the Chairman of the American Committee, the great majority of the countries circularised appointed delegates, to form a Preliminary Organization Committee. This has since been constituted the Permanent Committee of Organization of the Congress, and it numbers some 100 or more members, each country having a representation of not more than six. The Council of the Neurological Section of the Royal Society of Medicine have appointed the following English neurologists on this Permanent Committee, and they form the English Committee: Sir James Purves Stewart (*Chairman*), Dr. Kinnier Wilson (*Senior Secretary*), Dr. W. J. Adie (*Junior Secretary*), Prof. Edwin Bramwell, Dr. Gordon Holmes, Dr. C. P. Symonds.

After considerable correspondence Berne was definitely selected as the place for the meeting of the Congress, which is to be in session from Monday, August 31st, to Saturday, September 5th, 1931.

The next step was the appointment of a Programme Executive Committee, consisting of not more than two members from each of the Committees of the countries concerned. This Programme Committee met in Berne on August 29th and 30th of this year, under the chairmanship of Dr. B. Sachs, who with Dr. Henry A. Riley represented America. The English members were Sir James Purves Stewart and Dr. Kinnier Wilson. Nearly fifty neurologists attended the meetings of the Programme Committee, including the following: Austria, Prof. O. Marburg and Prof. C. Economo; Belgium, Dr. L. Van Bogaert; Czechoslovakia, Prof. L. Haskovec and Dr. H. Prochazka; Denmark, Dr. K. H. Krabbe and Prof. A. Wimmer; France, Prof. G. Guillain and Dr. G. Roussy; Germany, Prof. O. Foerster and Prof. M. Nonne; Holland, Prof. B. Brouwer and Prof. K. H. Bouman; Hungary, Dr. L. Benedek; Italy,

Prof. O. Rossi and Dr. V. M. Buscaino; Poland, Prof. J. Piltz and Dr. W. Sterling; Russia, Prof. M. Kroll; Spain, Dr. M. Prados; Sweden, Prof. H. Marcus and Dr. N. Antoni; Switzerland, Prof. R. Bing and Dr. C. Dubois. Roumania, Japan, and still other nationalities were duly represented.

Much important preliminary business was transacted by the Programme Committee. Dr. B. Sachs was unanimously elected President of the Congress, and the following eight Vice-Presidents, viz., Prof. Sir Charles Sherrington, and Profs. Bing, Kappers, Guillain, Marburg, Marcus, Nonne and Rossi. Among other appointments was that of Editor of the Transactions of the Congress, for which Prof. Brouwer of Amsterdam was nominated, with four colleagues, one for each of the official languages of the Congress, viz., Dr. Kinnier Wilson (English), Dr. G. Roussy (French), Prof. C. Economo (German), and Prof. V. Buscaino (Italian). After a great deal of discussion, owing mainly to the numbers of topics suggested by different countries, the following subjects were selected for consideration at the four chief sessions of the Congress:

1. Diagnostic and therapeutic procedures, surgical and otherwise, in brain tumours.
2. Muscle tonus: anatomy, physiology, and pathology.
3. Acute non-suppurative infections of the nervous system.
4. The rôle of trauma in the production of nervous symptoms.

In addition, it was decided that at least two afternoon sessions (and probably more) will be devoted to individual communications. For each of the four set discussions a Chairman was selected, and various names chosen as those of the opening contributors to them, each Chairman agreeing to be responsible for the final selection in respect of his own session. The essentially neurological character of the Congress was fully emphasized by the Programme Committee, and it was unanimously agreed to admit neuropsychiatric subjects to the programme list only in so far as they have a somatic basis.

As a mark of appreciation of their services to neurology a number of distinguished men were chosen to form a group of Honorary Members, included in which are Babinski, von Monakow, Pavlov, Ramon y Cajal, C. K. Mills, Dana, and Sir Henry Head.

The details of the hours of session, methods of raising funds, social aspects of the Congress, entertainment, publicity, were among other topics to which attention was directed. Messrs. T. Cook and Son were appointed official transportation agents for the Congress.

The meetings of the Programme Committee were characterised by cordiality and enthusiasm, auguring well for the success of the Berne Congress. Through the generosity of the American Neurological Association the whole of the members of the Committee, with their wives, were entertained to dinner at the close of the proceedings, and the spirit of international friendliness which then prevailed was particularly noticeable, amply fulfilling the hopes and justifying the aspirations of those with whom the idea of resuming such international conventions of the neurological profession originated.